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LECTURES

ON

PATHOLOGICAL ANATO

LECTURES

ON

PATHOLOGICAL ANATOMY

BY

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SECOND EDITION

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PREFACE TO THE SECOND EDITION

It is long since Dr Wilks requested me to prepare a new edition of his 'Lectures on Pathological Anatomy.' Those Lectures contained the results of careful observations, made by their author during the long period in which he had charge of the pathological department at Guy's Hospital.

But, in the mean time, great changes had been made in the aspect of pathological science, not merely through the addition of new facts, but by the general adoption, in a more or less complete form, of the views enunciated by Professor Virchow in his 'Cellular Pathology.'

My object was to preserve the general character and aim of the work, believing that it supplied a want very generally felt by students of medicine, as, indeed, the rapid sale of the first edition sufficiently proved.

But I found that the new aspect of pathology needed an altered mode of statement and arrangement of much of the valuable matter contained in the first edition; and also that new material was required, in the furnishing of which observations of my own were necessary, in order to maintain the original character of the Lectures; and these observations required time and labour, whose results are embodied in the new portion of the work, except the part treating of the Lung, which is still wholly due to Dr Wilks himself.

The principal sources of information employed by me were the records of Guy's post-mortem department during the time I had charge of it, and the invaluable 'Transactions of the Pathological Society.' I had, in my manuscript, referred to the inspections or specimens upon which the several statements were founded; but these so encumbered the work with voluminous references that it was thought necessary to dispense with them.

It was also, at first, my intention to have illustrated the descriptions by figures; but I found that drawings of diseased viscera showed little that is characteristic, without the aid of colour, which would have made the work too expensive.

My thanks are due to my friend Mr Thomas Eastes, M.B., for his kindness in helping me to revise many of the proof-sheets, in others of which the critic of orthography will find some faults, should he read as carefully as Mr Eastes would have done.

WALTER MOXON.

December, 1874.

PREFACE TO THE FIRST EDITION

HAVING been repeatedly solicited by the Students of Guy's Hospital to publish some Lectures which I delivered during the summers of 1857-58, on Pathological Anatomy, I have at length complied with their request, and now offer this book to their notice.

My original design, however, extended no further than to the publication, in the form of a syllabus, of some pathological tables, accompanied by references to certain typical preparations contained in our Museum; but finding that further explanation appeared necessary, I was induced to send to the press the whole of my lectures, in the exact form in which they were originally delivered. This circumstance will account for any peculiarity which may be observed in their style and arrangement.

The order in which the subjects are treated is not one which I should have chosen, but, since it is that adopted in our Museum, I found it convenient to adhere to it. The allusions to preparations have, of course, been mainly designed for the convenience of those Students and others who have access to our Museum; but they will not, I hope, be found to render the descriptions given in the text less intelligible to other readers.

The attempt to demonstrate the morbid anatomy of the whole body, in the few hours set apart in the summer session for a voluntary course, may require some explanation. It has always been my opinion, that every complete course of medical education should comprehend a systematic elucidation of the various morbid changes to which the human frame is subject, independently of the regular courses of lectures on medicine and surgery, and as a supplement to them; for, although it has been rightly considered by the

Examining Boards that the subject is included in these courses, since pathology is the basis of all true instruction in practical medicine, yet the subject is of sufficient importance to justify separate treatment. Moreover, it is well known that many experienced professors would gladly leave the demonstration of the structural changes of organs, in order to devote a larger portion of their time to the more practical matters relating to the symptoms and treatment of disease. This has always been felt at Guy's, and therefore, ever since that institution has possessed a medical school, it has been the practice of some of its most eminent teachers to deliver a course of lectures on pathology. The several licensing boards have recently shown their sense of the importance of the subject by instituting special examinations in this department; and it would be well, I think, if they insisted on its more systematic treatment by those in our hospitals who are qualified to undertake the task. Such teachers would then occupy a position in England analogous to that which in the Continental Universities is filled by some of their most distinguished professors.

To this statement, as to the purpose of these lectures, and my reasons for publishing them, I venture to add a word or two respecting my own personal pretensions. I do this with some reluctance, and solely for the satisfaction of any strangers into whose hands this book may fall; but it seems right to say, as a mere matter of fact, that I have for the last fifteen years made a daily study of the dissection of the dead, that I have now for many years held the appointment of Demonstrator of Morbid Anatomy, and that I have myself recorded between 2000 and 3000 inspections, of which we have an average at Guy's Hospital of more than 250 annually. I have also the charge of a splendid pathological collection. In the preparation of these lectures, I have sought to avail myself of the advantages afforded by so ample and varied a field of investigation, and have carefully abstained from making any statement unverified by my own observation and experience.

SAMUEL WILKS.

June, 1859.

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PATHOLOGICAL ANATOMY

DISEASES OF BONE

Malformation.—*Excess of development.*—Parts that are repeated in series in the body, such as the vertebræ and the fingers, are occasionally produced in more than their proper number. Examples of this are seen in these *supernumerary vertebræ;* for instance, here we have thirteen dorsal, each with a pair of ribs to it, and here are six lumbar. We need scarcely say that along with each additional vertebra all the soft parts which are connected with an ordinary vertebra are present, so that there is rather an additional zone to the body than a mere excess of bone development. The same remark will apply more or less obviously to the other bone malformations.

When an additional vertebra is present in the lumbar region it is, in some cases, difficult to say whether the extra vertebra is a lumbar or an additional sacral piece. Thus, in this specimen, when looked at in front, it appears as if an extra piece had been added to the upper part of the sacrum, but from behind the arches and spine are seen to be distinct, like those of the lumbar vertebræ. Sometimes when there are but five lumbars, the fifth may coalesce by one or both of its sides, with the sacrum below.

Supernumerary ribs are short processes seen in the neck and loins, and called cervical and lumbar ribs. We may here also show you this specimen of *episternal bones,* where two small tubercles are seen growing on the upper edge of the sternum; they are thought to correspond to the coracoid bones of birds. Also this humerus, exhibiting a *supra-condyloid process,* which formerly was catalogued as an exostosis. This process, which is situated about two inches above the inner condyle, forms a hook, and, when a ligament extends from its extremity to the condyle below, a hole or ring is produced corresponding to that supra-condyloid foramen met with in the humerus of the carnivora and some other classes of animals, which serves to transmit the median nerve and brachial artery. You will find many specimens of *supernumerary fingers and toes* on our shelves. These do not always contain bone, and when bone is present it is often only an excrescence of the bone of the next finger or toe. This multiplication affects chiefly the terminal phalangeal bones, the metacarpus and carpus, &c., being less liable to be implicated, though sometimes the cuneiform bones, &c., may alone be found in too great number.

Deficiency in development.—A remarkable form of this is seen in the *anencephalous* fœtuses, of which we have numerous examples, and in which, while the brain is absent, the bones of the cranium are scarcely developed. There may be a deficiency limited to parts of the skull, as in this preparation, exhibiting the want of a portion of parietal bone; sometimes such deficiencies are large, but the most interesting cases of the kind are exemplified in this specimen, where there is an opening in the occipital bone, through which a portion of brain or of its membranes protrudes, constituting an *encephalocele.* These are not always occipital, they may be frontal, and occasionally are lateral; they do not usually pass through sutures. In some rare cases such protrusions come through the base of the skull, and may project into and from the mouth in very strange forms.

A still more common deficiency is a want of union of the posterior arches of the vertebræ, constituting *spina bifida.* Occasionally the bodies of the vertebra may be split or double. The sternum may be cleft, and so may the hard palate, through a failure of union in the anterior arches, a failure which corresponds to the non-union of the posterior arches in spina bifida. The failure of union of the posterior arches may affect the whole cranio-vertebral system, the arch of the skull with the brain being absent, as well as the arches of the spinal vertebræ. In other examples hydrocephalus coexists with the spina bifida. The non-union varies in its extent in the spinal column; some cases show an open canal the whole length of the spine, while others exhibit a deficiency limited to a particular locality. This is generally in the loins, where we find the posterior arches of two or three vertebræ wanting, and the membranes protruding in the form of a sac. This sac is generally about as large as an orange, and usually consists of the spinal membranes filled with fluid, protruding outwards through the opening in the vertebral canal; its inner surface is then lined by the visceral arachnoid, and, therefore, the fluid within it is the sub-arachnoid fluid, and communicates directly with the fluids in the ventricles of the brain. This visceral arachnoid is closely adherent outside to the parietal arachnoid and dura mater, and the latter in their turn to the integument, so that it is difficult to separate these structures, which form the wall of the sac. The medulla or cauda equina, at the spot where the bones are deficient, may continue its way downwards as usual; or if the opening be large the nerves may pass into the sac, and be distributed upon its walls. The nerves go regularly from their points of origin on the cord; some run a short distance on the outer wall of the sac, curve round, and then go through its midst to their exit at the fore border by the intervertebral foramina; others make longer windings on the outer wall before they take their forward course, in which they sometimes stretch back to the cord; on reaching the intervertebral foramina they pass through the dura mater in two rows, and make their ganglia as usual. Spina bifida is not always of this simpler form, which is termed, from its components, *hydromeningocele*. In another form, which is not altogether rare, the central canal of the cord is enormously distended with fluid, and the cord itself is thus expanded into the tumour, constituting *hydromyelocele*. These conditions are analogous to common hydrocele in being irritative dropsies; they probably depend on irritation of the nervous centres in fœtal life. A funnel-like depression or umbilicus in the middle line of the tumour will generally signify the point of insertion of the spinal cord on the wall of the sac.

A simple *want of union*, without protrusion of the spinal contents, is sometimes met with, as in this specimen of atlas, where the two halves are perfect, but are not united, though probably in the fresh subject they were joined by ligamentous tissue. Sometimes one half of the arch of a vertebra is not developed, and thus the spine bends towards that side and a distortion results. This specimen is a very interesting and remarkable example of such an occurrence. You will see that the spine is distorted from a fusion of three dorsal vertebræ, arising apparently from this want of regular development; and in the neck you will also see that the arch of one vertebra is not completed, but that one half is ankylosed to the arch of the vertebra above, while the other half remains free, and alone gives origin to the spinous process. The spinous process, too, may be sometimes *bifid*. Absence of certain bones is met with in cases of deficiency in the number of the ribs; or in cases where the hands and feet are articulated to the scapula and pelvis respectively; or in cases of imperfectly formed or absent fingers and toes. Such deformities are sometimes hereditary. The man from whom these models of the hand with deficient fingers were taken stated that the malformation had occurred in some members of his family for five generations, but not always in a direct line of descent. Among other abnormalities might be mentioned *bifid* ensiform cartilage, which sometimes produces a foramen in the end of the bone, bifid ribs, bifid odontoid process of axis. *Ossa triquetra* will be hereafter mentioned.

Irregular development of skull.—Less striking, but more really important than these obvious variations of bony development (which, as we have said, go with an equal extent of malformation in the corresponding soft parts), are certain irregularities in the normal proportion of development in the several bones of the skull. In normal skulls the several bones bear a nearly constant proportion in size to each other, but sometimes one or more of the bones of the base is too small, and Virchow has shown that the surface growth of the several bones is dependent on the persistence of the sutures, too early a coalescence of a suture checking the growth of bone that should occur along it. This *premature synostosis* may be limited to a single suture, while growth occurs at the others; in this manner the shape of the skull, and with it the development of the facial bones, may acquire characteristic modifications. Synostosis of the sagittal suture stops the widening of the skull, and meanwhile it may grow disproportionately long, or *scapho-cephalic*; synostosis of the coronal suture checks the lengthening, and allows the widening, thereby producing a *platy-cephalic* skull; other imperfections in cranial form will readily be traced from other seats of the synostosis. It is a most interesting question whether these changes in the bone are primary, and give rise to those imperfections or irregularities of the brain which must conform to them; thus a premature synostosis of all the sutures necessarily creates a small head or *microcephalus*, and this is found often enclosing the small brain of an idiot. It is thought by some that this closed smallness of skull causes the smallness of brain by preventing its development. Others have shown that microcephalic skulls are not always synosteal. Thus, Dr. Down, from observations of the skulls of two hundred idiots, concludes that the deviations of the cranium have been rather the sequence of circumstances arresting the development and growth of the brain, and not the result of premature ossification. Extreme microcephalus is found without any ossification of cranial sutures. In the skull of a *cretin* described by His the base was short, and yet its bones were so far from being synosteal that they came apart on maceration, though the subject was fifty-eight years old. The whole skeleton, as usual in cretins, presented puerile characters. Whether in natural development the bone moulds the soft parts, or the soft parts the bone, is as yet undecided, but abnormal changes in either no doubt influence the other. Instances of this we shall presently see.

Atrophy.—The simplest condition is that arising from *disuse*, a good example of which is seen in this skeleton of a man who, being paralysed in his lower extremities, used his arms for progression; the bones of the leg, you see, are much wasted. After any *fracture* of a bone, which produces injury to its nutrient artery, a partial wasting of the fractured ends may take place. Violent *separation of an epiphysis*, which is a form of fracture far from uncommon in early life, often, though not necessarily, leads to deficiency of development of the fractured bone. The bones, in cases of *congenital paralysis*, share the general smallness of the affected limb—a condition, however, which is rather a result of undergrowth than atrophy. The bones waste in old age, the rind of the bone becomes thinner, brittle, and porous, and the medullary canal enlarged and filled with a fatty matter. The effects of *age* are also seen on the neck of the thigh bone in persons of advanced life, in whom the neck sinks to a more acute angle with the shaft; it is also shortened, and of open cancellous structure; its condition requiring care in distinguishing it from the results of fracture. A somewhat similar atrophy to that often seen in old persons is met with in limbs which have long been inactive from disease of the joints, or from the patient being bedridden; the bone is found to have a thin shell, and the medullary canal is filled with a soft yellow matter, consisting almost entirely of fat; the cancellous structure of the bone is also occupied by it. Atrophy may also arise from *pressure*. This is especially seen in aneurism of the aorta, where the tumour, by constant pressure against the spine, gradually erodes the bone until the bodies of the vertebræ are destroyed, and in some severe cases the canal laid open; the intervertebral cartilages remaining comparatively unaffected. The loss of substance is a simple destruction of the bone, and in nowise can be called ulceration or caries. In the same way the sternum, ribs, &c., may be affected, as may the long bones from the immediate pressure of aneurisms. Sometimes the bone appears lifted and expanded before the advancing aneurism. Thus, parts of the sternum and clavicle may rise like an incomplete shell before an aneurismal tumour, so, perhaps, making it difficult to distinguish the aneurism from a pulsatile growth expanding the bone. It seems improbable that the bony matter thus apparently lifted is really the natural bone, for this is not extensible and ductile; we must suppose that, the original bone being removed, a growth of bone is produced in the periosteum, as this is thrust before the advancing tumour into new positions and shapes. In the calvaria you may often notice deep depressions along the sides of the longitudinal sinus, produced by the pressure of the *Pacchionian bodies*, and *tumours* of all kinds may produce like effects on the bones.

Apart from these manifest causes, you may often find thinning of the bones; thus in the cranium, and especially in *maniacs*, this condition may be seen, although in some of these the skull may be very thick, and in others very thin, or it may be thick in places and thin in places. Sometimes, on holding up a calvaria to the light, you may see it almost as thin as paper on each side of the median line; and sometimes, as in this example, there is a depression on each side, and a raised ridge of bone along the temples. In this skeleton, which is evidently affected with *rickets*, the cranium, you will see, is remarkably thin, and this fact is, we think, compatible with the statement that hypertrophy may result from rickets, for in this case the patient is young and the bones are soft; whereas, if the expanded porous bone had become filled with a hard bony material, an enlargement would have resulted. There is another remarkable atrophy of the bones of the skull which is not very uncommon, but yet which we do not remember to have read of in books; it is an extreme thinning of the *petrous bones*, orbital plates, and adjacent parts of the base, whereby they become quite diaphonous, and the point of the scalpel may be easily made to penetrate them. It is a condition accidentally met with, and does not indicate, as far as we are aware, any distinct pathological state.

The thinness of the orbital plates must be remembered in cases of penetrating injuries about the eye, for occasionally these plates are broken by slight causes, such as a thrust with a tobacco pipe, and this when there is no external sign of the severity of the injury; perhaps, too, for a time there may be no symptoms, even when the brain is subsequently found to have been penetrated.

Hypertrophy.—*Relation to inflammation.*—Low augmentative processes are registered in the bones more obviously and enduringly than in any other component parts of the body, because of the permanency of the bone produced. Very slow augmentative processes of low intensity produce increase in the size of the bone without giving rise to the heat, pain, &c., which constitute clinical inflammation. These are called by the general name *Hypertrophy.* When the cause of production is somewhat more intense, or acts with greater rapidity, the vascular disturbances of inflammation attend the hypertrophy; and when the intensity is yet greater, the bone ulcerates, softens, or perishes in larger measure by necrosis. Considering this series of changes only anatomically, so that the heat, pain, &c., are out of recognition, it will be clear to you that hypertrophy, the simple, slow augmentation, cannot be *anatomically* distinguished from the milder inflammation which is characterised by mere vascular disturbance without destruction of the bone, and with new formation upon or in it. But the higher inflammation, resulting in ulceration, caries, or necrosis, stands plainly distinguished from hypertrophy, and is called by the name *inflammation.* It is necessary to clearly understand this relation of the word inflammation to the anatomical facts we are considering. Inflammation is the name of the process of which hypertrophy, caries, and necrosis are the anatomical results; but the process which results in hypertrophy is often latent and unrevealed by symptoms, so that hypertrophy then exists without evident inflammation. On the other hand, caries and necrosis are always accompanied by the clinical phenomena of inflammation, so that they are commonly and conveniently called inflammation. It is very

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spontaneous or non-inflammatory increase of bone, which is called by the general name of hypertrophy, offer us an actual enlargement of the size of the bone without increase of its density; while others, on the contrary, show no superficial enlargement, or but little, while the density and closeness of the texture of the bone is increased through the filling up of Haversian spaces, until the bone resembles ivory. Now, most cases of hypertrophy of bone include both these kinds of increase—the superficial and the interstitial. When the increase is in density only the name *sclerosis* was given by Lobstein. The ultimate condition of the bone is, no doubt, much the same in all hypertrophies, whether they arise from inflammation or not; and thus in this piece of femur (which may be in reality from a case of ostitis) the structure is very dense and like ivory, as it is also in this slice of an hypertrophied cranium, which is probably not inflammatory. You might think that a bone presenting the ivory-like condition must be very different from an ordinary one; but, although it may be so to outward appearance, the microscope does not show much except to a practised eye. The greater compactness of *old* bone arises from a filling up of the spaces in the bone with concentric bony laminæ.

But formations of bone sometimes arise on the surface by ossification of the periosteum. The method of this difference lies here, that, when the bone enlarges on its surface, the new bone is produced directly *from the periosteum;* whereas, when the bone condenses without enlarging, the new bone is formed *from the medulla* lining the cancelli.

In either case the bone formed has the general characters of osseous tissue (*tela ossea*), with which you are familiar, and it passes through its proper developmental stages. Thus, when young, the new bone on the surfaces of the old is spongy, being formed of spicules mutually uniting to make a meshwork, a primary cancellus, in which the unossified remainder of periosteal tissue is seen supplied in each mesh by a vessel. Then the ossification of this periosteal tissue proceeds until the vessel is surrounded by close bone. After some time the formation of medullary spaces will occur in the deep part of this new dense bone, and so a cancellous structure again returns. But this secondary cancellation is not the same as the primary, for its meshes are occupied by marrow and not by unossified periosteum as the first meshes were. So you will see that fresh-formed bone is less organised, and is wanting in adaptation of its parts to the situations they are to occupy, the surface of the new bone and its deeper portions being much alike; but, as in all other permanent formations, in process of time the new part tends to assume the characters of structure and arrangement proper to the healthy tissue, and so the surface becomes dense and the deeper part cancellous. It is thus that outgrowths of bone acquire even marrow canals, and in like manner the marrow canal is modelled out through the callus of badly united fractures. The knowledge of this will enable us often to infer approximately the oldness of a bony hypertrophy, and to understand why some adventitious bone is close-textured, being new, while other is cancellated, being old. Notwithstanding this, we cannot always explain the differences of solidity in new bone as due to the stages of development. Indeed, the ivory-like "eburnation" of some new growths attains to a degree of density much greater than that of the ordinary close-textured, newly formed bone. This is especially true of the ivory exostoses of the cranium and orbits.

General and local hypertrophy; varieties of hypertrophy.—So different are the appearances and the clinical nature of these bony overgrowths, that it is difficult to distribute them into varieties for description. They are usually divided into *general and local* hypertrophies, though the distinction is not very definite. Still viewing at large the cases of hypertrophy, we see that, in some, the change affects many bones, or even all the skeleton, while in others the increase is limited to one bone, or even to the small part of a bone.

Let us first consider the more *general* forms of hypertrophy—we mean the more generally diffused overgrowths of bone. A convenient term for these is *Hyperostosis*. It will correspond with the old and accepted use of this term, if we mean by it those enlargements of bone that are less circumscribed upon the bones they affect, generally implicating the whole bone, or even many bones. More circumscribed formations of bone are called *Periostosis*, and if these are so defined as to resemble tumours they are called *Exostoses*.

The simplest form of general enlargement, or *hyperostosis*, arises from increased function, as seen in the case of that same skeleton of the paraplegic man who used his arms for progression, which we showed you just now. It is perhaps hardly fair to call this hypertrophy; it rather is the state of bone corresponding to thorough muscular development, and can scarcely be construed into morbid anatomy. Quite different is the case with such bones as these; they are specimens from the skeleton of a man sixty years old, affected with general hypertrophy of the bones; observe that the surface is porous and has a singular mortar-like look, being whiter than natural, and not having the usual surface-structure of bone. Dr. Goodhart, who made the inspection, says, "When fresh the periosteum appeared natural, but the bone was pink throughout, and though firm and heavy, much softer than normal bone." "Those bones that could be examined, as the cranium, ribs, pelvis, thigh bones, &c., were affected, but apparently not the bones of the face. The left clavicle, for instance, was two and a half inches in circumference, and on section showed a uniform surface, with hardly any medullary cavity and no cancellous tissue. Section of the femur showed two parts of the bone distinct from each other, one being the old compact bone immediately around the medullary canal, and external to this a good thickness of more vascular new bone." The femur weighed 2 lbs. 14 oz., and was from six inches and a half to eight inches and a half in circumference. The pelvis partook of the form usual in rickets. The microscope showed that the diseased bone had undergone a considerable change; the Haversian canals were enlarged and of irregular shape. During life the bones were observed to enlarge for twelve years, and the man died of fixation of the chest through disease of the ribs. This porous form of general hyperostosis is called *osteoporosis*. The loose, light, porous condition of this bone, which is the femur of the case in question, will strike you as very remarkable. There is none of the usual hard bone of the shaft; it is all like open sponge.

Progressive enlargement of the skeleton affecting some parts more than others, and this so as to cause tumour-like masses in places, has occurred in several recorded instances. In Dupuytren's case the growths began in a sucking child at many points in the trunk and extremities, and continued enlarging until death. The adjacent ends of the femur and tibia grew to the size of "knobby potatoes," while other bones were less affected and others natural. In Saucerotte's case a man, thirty-nine years old, grew in four years 59 lbs. heavier, while his flesh wasted away, his head grew too large for ordinary hats, and his eyes stood out through pressure to a level with his forehead.

Whether these cases are all of the same nature, and if so, what is that nature, are questions we must yet hold doubtful; it is natural to ask after an association with syphilis or rickets as causes, for there is no doubt that either of these disorders will give rise to enlargement of bone. There is, however, no sufficient answer to these questions. Syphilis has generally not been present; but in some cases of general enlargement of bones, some of the bones, say the tibiæ, are found curved as in *rickets*. Take this instance from a case of porous enlargement or *osteo-porosis;* it is probably of the same kind as the first we mentioned, but its history is deficient. In this case all the bones of the skeleton are much enlarged; you will see that the sections display a cancellous structure, and the bone, instead of being compact, has the appearance of mortar. This is especially well seen in the skull. The grooves for the meningeal arteries are very deep. Now, the tibiæ have ricketty bends, so as to suggest that such a disease has given rise to the change. In the subject that furnished this other specimen, where all the bones were enlarged, there was a distinct history of rickets; also, in the first case of osteo-porosis we described, the pelvis, you remember, had the ricketty form. In a case related in Virchow's 'Archiv' two brothers were affected with general hyperostosis.

Remarkable examples of hypertrophy are frequently met with in the removal of the *calvaria*, where the bone is often found twice its usual thickness; the increase may be uniform, or greater in some parts than others. This thickening of the cranium has also been ascribed by some to rickets; while others, with Virchow, ascribe this view to a total misconception of the nature of rickets. We will speak subsequently of this question. If we had any knowledge of the nature of that general progressive porous enlargement of the bone or osteoporosis, of which we have given you cases, we might probably find these thickenings of the skull to be of the same nature, but less extensive. The peculiar porous appearance of the bone is generally present in them; but, unfortunately, there is no history in connection with the cases.

Another very terrible form of enlargement of bone is a hyperostosis, generally limited to the bones of the *skull and face*, increasing them enormously, so that in Fourcade's case, figured by Lebert, the macerated skull weighed 8¼ lbs., the lower jaw 3 lbs. 6 oz., the whole, and especially the facial aspect of the skull, being horribly deformed with crowded and heaped and mostly rounded outgrowths, very hard, and not at all resembling the porous condition just described.

From the lion-like look and the real likeness of the disease of the bone to the hypertrophy of soft parts of the face, which constitutes the kind of leprosy called "Leontiasis," the name *Leontiasis ossea* has been given to this disease. It has been found associated with a similar condition extending to other bones of the skeleton, but, on the contrary, we more frequently meet such an overgrowth limited to one or two bones of the skull or face, or to parts of a bone. Thus, the *sphenoid* or *malar*, for instance, may grow into tumours; or more frequently the jawbones swell generally, or at a circumscribed part, producing enlargements which, however, partake more of the nature of tumours. Some of these, implicating for the most part the walls of the frontal sinuses or of the antrum of Highmore, are found exceedingly hard like ivory, and are known as *ivory exostosis*.

Under the name of *Osteophyte* one would widely include every sort of new growth of bone, whether hypertrophic or inflammatory.

This term, indeed, is used generically, and would include the kinds of general hypertrophy we have hitherto dealt with, but it commonly is applied in a more limited signification to name *circumscribed additions to the surfaces* of bones. It was believed by Lobstein that these superficial formations or osteophytes represented the ossified blastema from various sources, which their forms would indicate. The diffused osteophyte coming from under the periosteum, the network-like from periosteum itself, the styliform from the tendons and aponeuroses, and the cauliflower-like from the interstitial tissue.

These names are not now much used, but if you look at many of the bones in our museum you will see plenty of examples to which they would be applicable. Thus we may mention a very common affection of the spine, in which new bony matter is found on the bodies of the vertebræ, uniting them together. More or less of this condition is found in a very large number of the bodies we daily examine, especially in those of hard-worked men. Some have called this condition *ankylosis* of the vertebræ, or ossification of the ligaments, but what we find in the first instance is an ossification of the surface of the intervertebral substance between several of the bones on one side, whereby slight bony projections are produced, the ossification not extending deeply into the fibro-cartilage. At a further stage these several exostoses may unite on the bodies of the vertebræ, and thus a mass of bone is seen lying on their surface resembling a thin lath of osseous material fixed upon the spine; or, sometimes, as if a quantity of soft bone (as it were mortar) had been poured upon it and then hardened. We often find this on one side alone; and if the spine be curved, on the side of the concavity. There is a variety of osteophyte called by Rokitansky *puerperal osteophyte*, of which we have mounted this specimen as an apparent example. He states that during pregnancy a new deposition of bone takes place on the inner surface of the cranium especially, seen as a slight layer along the sides of the longitudinal sinus, and also in patches on other parts of the calvaria and base. This specimen, from a young woman who died at the fifth month, shows certainly a new layer in the form of slight elevations along the upper surface of the skull and on the frontal bone; but we question very much whether it is related to the puerperal state. There must be much uncertainty of this until more extended observations are made, for such irregularities are constantly met with on the inner surface of the skulls of persons dying from a great variety of diseases.

A case is recorded of bony growth both within and without the skull which augmented at several successive pregnancies, and the puerperal osteophyte is said to occur on the outer surface of the skull and facial bones. Virchow thinks it is like puerperal endocarditis in its relation to the puerperal state. We have no experience of it.

The prominences of bone which surround joints in the state called *chronic rheumatic arthritis* are strictly osteophytes, though the definite disease they arise from removes their consideration to its own place. All superficial inflammatory formations of bone would likewise deserve the title, but where any known cause of inflammation has produced them, their consideration is merged in that of the inflammation itself. If produced by more recent and more acute inflammations, as in caries, these osteophytes are spongy and spicular. In chronic rheumatic arthritis they are rounded and dense.

When an osteophyte *forms a bony tumour* on a circumscribed part of the surface of a bone it is called an *exostosis*. This term is given when the growth is of such a prominence as to pass beyond the proportions of a mere bump or elevation. Such elevations as are mere bumps are called *nodes;* and the distinction generally corresponds to a real difference of cause, for nodes are mostly traceable to injuries or to the neighbourhood of ulcers, or to ossification of old syphilitic periostitic patches; whereas, on the other hand, exostoses are generally of unknown origin, and correspond more to the nature of tumours to which their surgical treatment practically allies them.

The distinction between an exostosis and an ordinary osteophyte is not very defined; speaking generally, any diffused new bone, on and distinct from the old bone, would be an osteophyte; but an outgrowth of the surface, not showing a distinct demarcation, would be an exostosis. Exostoses are of various forms, and may be met with as projecting processes on the long bones, as seen in many specimens in the museum. They, however, are not indifferent in point of situation, but have certain seats of preference. Thus a very frequent position is the end of the great toe; an exostosis often occurs in young people as a round tumour attached to the end of the last phalanx; a section, as in these specimens, shows that it is developed from cartilage, the latter forming its circumference, while the centre contains the bone or ossified portion. Such tumours afford good opportunity for witnessing the process of ossification from cartilage.

Exostoses on the skull have often a deep furrow round their base. Exostoses may occur as isolated rounded growths, perhaps of considerable size, on the outer or inner surface of the cranium, and if the latter they may produce important symptoms of brain irritation. They may hold any situation indifferently, but we think attention should be specially directed to those which form on the inner surface of the skull, chiefly on the frontal bone around the crista galli. These are not very uncommon, and are found accidentally as one or two little growths, like small mussel shells. Their number may be greater, and their form less distinct, until the whole interior of the frontal bone is roughened with projections, or the same state is found to extend to neighbouring bones, or even to the whole interior of the skull, reminding us of the nodular hypertrophy of the facial bones before mentioned. In these latter severe cases epilepsy or idiocy has accompanied the condition.

We have not been able to learn whether the lower degrees of this state of frontal exostosis are associated with family tendency to cerebral affection; but these lower degrees in the form of small shell-like growths with constricted bases coming often symmetrically from the frontal bone near the crista galli are far from uncommon.

The larger growths, isolated and often nodular on the surface, appear to go inwards through the dura mater; but this really extends very thinly over them. They are hard to tell from bony growths *in* the dura mater, which we shall presently speak of. The gathering of the intra-cranial exostoses around the region where the sinuses are late in developing, and where, therefore, unusually protracted activity is manifested, would suggest this greater activity as a cause of these growths; and in support of such a view we have the frequent occurrence of exostoses of the long bones at points corresponding to the epiphyseal cartilages where activity of growth is continued long.

Inflammation.—In speaking of hypertrophy of bone we have followed the common idea of the dry hard bone of the skeleton. This simple notion of bone suited very well for the description of the various kinds of augmentation and alteration of its form which constitute hypertrophy; but it is most important now that we come to consider the more violently active changes in bone to remember that the hard bone of the skeleton is only part of entire bone; indeed, *the bone of the skeleton is but the skeleton of the bone.* In a living bone the hard osseous tissue, or *tela ossea* proper, is covered outside with periosteum; the cavities or cancelli within it are filled with medulla; vessels, either arteries or veins, permeate the substance running in the Haversian canals; even the lacunæ are occupied by cells, whose processes stretch in the canaliculi to communicate with the contents of the lacunæ around.

A living bone contains all these soft parts, and its life is maintained by them. Further, it commonly also is furnished with cartilage at its articular extremities, whilst ligaments, tendons, muscles, or fat are more or less intimately united with its surface, altering not only the periosteum but the texture of the true bone at the points of union, so that if you will carefully consider the surface of any bone, say of this femur, you will soon learn to distinguish easily those parts which have been in continuity with the several kinds of tissue which enclose the femur. Here, where the fleshy muscle took its rise, here, where the gastrocnemius tendon was attached, and here, where the adipose tissue lay in contact with the bone about the joint, you can discern altogether different kinds of surfaces which you will find then quite characteristic, so that in this way you can tell by examining the bone whether flesh, tendon, or fat was connected with any given part of its surface. But the most important connection of the hard *tela ossea* is not this anatomical connection with the parts around for the purpose of supply; it is rather the historic or physiological connection which it has with the soft parts, in that the bone arises from the cartilage periosteum or medullary tissue (ultimately nearly all from the medullary tissue), and it is always ready to return into the soft state again. The dry bones we are familiar with all our life impress us with a natural but false idea that the bone is a permanent and passive sort of thing, having no proper vitality or versatility.

The deepest layer of periosteum close down upon the bone (at least in growing bones if not during the whole of life) presents characters closely resembling the tissue of bone itself. The cells of the periosteum have structure and arrangement resembling those of bone lacunæ, and only need impregnation with earthy salts to constitute true bone. In this state of transition the deep periosteum is called *osteoid tissue*, and is, in fact, young bone, for it is by this simple transformation of the deep periosteum into osseous tissue that the bones grow in thickness. A corresponding but reversed activity is normally present in the marrow cavities, for as the bone increases outside the marrow cavities form in the older inner texture. These cavities arise thus: the calcified tissue loses its lime in the area that is to be a cavity, and the lacunæ-cells, set free from the lime, multiply, producing numerous small cells in their place, many of which closely resemble the cells of lymph or white blood-cells, but they generally have a reddish colour. Some of these cells may take up fat to form the adipose tissue of marrow. A fresh interest attaches to this medullary tissue since the arguments and observations of Naumann, by which he endeavours to prove that the reddish lymphoid cells are really young red blood-cells, and that the red blood thus is formed in the marrow of the bones.

In the multiplication of the lacunar cells some of them show the following variety:—The nuclei of these cells multiply within while the cells grow, and are then found very large and containing a number of nuclei. The large polynucleated cells so formed are called myeloid cells, and are very characteristic of developing marrow. We shall presently see their relation to certain bony tumours. We mention all this because it is necessary that the history of bone formation should be borne in mind, or we cannot follow and comprehend the various results of inflammation in bone. By dwelling on the natural history of bone formation we are taught to regard bone as very far different from the mere petrified lever or shield it is naturally at first thought to be.

True it is that, while the vessels and lacunar cells of the bone are encased and fast set in stony substance, the vital processes must be very restricted, but now that we know, first, that each lacuna contains a living cell, and second, that this living cell is capable of so far ruling over the domain around as to determine the absorption of the lime-salts, thus setting itself free from its stony prison, we recognise that the tissue of bone in the matter of inflammatory and other action has only a temporary disadvantage in comparison with other tissues. Its forces are slow to mobilise, so that it may be overwhelmed in a sudden attack of inflammation, because its cells and vessels are not free to exert those powers of accommodation which would make their activity equal to the emergency. But if the inflammation is less severe, so that time is given for the change the bone is to go through, a very interesting process takes place which, better than any other thing, serves to prove the individual activity of bone-cells. If you look at these microscopic sections of inflamed and necrosed bone, you will see that the outlines of the Haversian canals of the solid and of the trabeculæ of the spongy bone are not straight as they should be, but are made most irregular and eroded-looking, through the hard bony surface being pitted deeply with little pits called *Howship's lacunæ*. These pits are filled in the fresh and living bone with a soft granulation tissue; they often sink deeply into the bony surface.

Now, if you compare these pits with the lacunæ you will see that each pit is in the position of one of the lacunar territories, so that no doubt will remain that each pit represents the area around the lacunar cell which has had its stony salts dissolved out.

The limitation of this solvent action to the figures of the lacunar territories will leave no doubt that it is to the activity of the lacunar cells that the solution is due. Our knowledge of this activity of the lacunar cells is due to Virchow, who has shown how the *tela ossea* actively works in the inflammatory process. The lime-salts are removed from around the bone-cells, forming thus the Howship's lacunæ; and the bone-cells, set free, proceed to multiply, and so produce a quantity of new tissue, which is like ordinary granulation tissue. We think that so much as this is fully proved, and that the granulation tissue thus formed may either perish in the spread of the inflammation, or, under favorable circumstances, repair the disease by forming new bone or fibrous membrane; but we think it is not shown that pus is so formed; it is probable that the progeny of the bone-cells only tends to repair, and not to pus formation. It might naturally be thought, and has been thought by some, that pus can dissolve bone. This idea seems favoured by the spread of abscess in and through bone, as though by liquefaction of the bony tissue. But it has been proved by experiment that pus has no solvent power on bone; and it is agreed that the spread of suppuration in bone is a vital process in which the living cells of the bone are the essential agents. If the bone with its cells die no solution of it occurs, but it must then be cast out as a dead body or "*sequestrum*."

The inflammation of bone produces results like those of inflammation in other structures; but the hard inextensible character of the *tela ossea* of course prevents any rapid accommodation of its tissue to very great vascular changes. Through this unyielding nature it is unable to go through intense attacks of inflammation, and consequently such severe attacks very frequently cause death of the portion of bone that is implicated in them. Death of bone is called necrosis, and it is, indeed, a very frequent result of severe inflammation of bone, but otherwise we find the inflammatory process in bone to have degrees and varieties corresponding with the degrees and varieties observed in inflammations of the soft parts. Thus, inflammation of bone is chronic or acute, plastic or suppurative; simply ulcerous; unhealthily ulcerous, then called *carious;* or gangrenous, then called *necrotic*.

The active inflammatory changes take place in the soft parts of the bone, its periosteum, or its medullary membrane. In some cases both these are implicated, in others only the one or the other. When both are involved, and thus the entire bone is included in the scope of the inflammation, it is called by the general name *ostitis;* when the action is limited to the periosteum it is called *periostitis;* when limited to the medullary membrane, *Osteo-myelitis*. This latter affection may follow the extensions of the medulla throughout more or less of the loose cancellous texture of the bone.

Periostitis.—The periosteal membrane may become inflamed from an external cause, as a blow, or from a constitutional cause, as scrofula or syphilis. The membrane becomes highly vascular or hyperæmic, and after a short time an exudation occurs in and beneath it, by which it becomes swollen and soft, and so is easily stripped off the bone; at a further stage lymph is formed, and this is, for the most part, beneath the membrane, and if circumscribed constitutes a node. This new product may be absorbed and the part return to its natural condition, or it may undergo further development, especially if a large part of the surface is affected, until a diffused layer of it may become bony, and be connected to the shaft by blood-vessels. Thus, an additional layer of bone is added to the old, the periosteum itself being somewhat thickened. The result of this process will be seen upon many of the bones in the museum. It appears as a distinct new layer added to the circumference of the shaft. A small mass of lymph is, however, generally absorbed. You might naturally inquire whether the inflammatory product, which thus ossifies and adds to the size of the bone, is yielded by the bone or the periosteum; no doubt the chief part is produced by the periosteum, which is much more rich in vessels than the bone it covers. Experimental transplantation of the periosteum into the soft parts has proved its tendency to form bone; any piece of periosteum, except pericranium, when it is successfully engrafted in subcutaneous tissue changes to bone, passing often through an intermediate stage more or less perfectly cartilaginous.

Any displaced periosteum in cases of fracture or abscess ossifies, while in cases where portions of the periosteum slough no bone is formed at those parts. This might suggest that ossification is limited to the periosteum, yet it has been proved that the ossification which commences in the periosteum will extend to the tissues around, and that not only to the fibrous tissues, but sometimes even to the muscles. Thus we become aware that periosteum and parts around will certainly give rise to bone. But it is equally certain that the bone itself, *i. e.* the medullary tissue in the cancelli of bone, will produce new bone. This we see in the ossifying granulations which spring from the exposed articular ends of bone in ankylosis of the joints. Such ossification from the medullary tissue is sufficiently evident also in the common process of condensation of the bones, which is nothing else than ossification of the products of the medullary tissue lining the cancelli. Hence we may conclude that the ossific new product in periostitis is yielded chiefly by the periosteum, but partly by the bone. In the case of the pericranium it is chiefly from the bone itself that the new bone comes, for the pericranium is much less vascular than ordinary periosteum, while the bones of the skull are rich in vessels.

Periosteal abscess or acute necrosis.—When the inflammation is extensive and severe, pus forms under the periosteum, and raises that membrane off the bone to form an abscess around the bone. Thus arises a terrible and very dangerous disease, which is not very uncommon; it nearly always occurs in children or young persons, and is usually ascribed to some injury which is generally not severe. The danger that attends it arises from its disposition to set up a very grave form of pyæmia, during which abscesses occur in the heart and kidneys much more often than in pyæmia from any other cause. The disease is not usually limited to the periosteum; indeed, some French pathologists have said that necrosis of the bone does not occur unless the medullary membrane is implicated. Certainly section of the bone usually reveals an inflamed state within; lymph and pus are found within the medullary cavity and cancelli; yet in many cases this is not so, but the bone is dead, while the interior exhibits no signs of former inflammation. Sometimes the disease kills very rapidly, as, for instance, in six or seven days; indeed, before pus has time to form in large quantity. The periosteum is then found detached and the bone separated from it by a dirty, brownish, turbid fluid, consisting of pus in which some altered blood is mixed. If the patient live longer, say for two or three weeks, a large quantity of pus is found around the bone, the bone itself being quite naked and white, or, more strictly, of the colour of milk of sulphur. The disease generally stops at the epiphyses, but not always; it very rarely, however, reaches the joints. It nearly always attacks one of the long bones, more generally of the lower extremity, but it has been known to affect the pelvic bones, pterygoid process, &c. We have seen it limited to the middle phalanx of one finger in a case rapidly fatal with the characteristic pyæmia. Fortunately, however, the disease is not always of this great severity. It may be only less rapidly fatal, lasting six weeks or six months; but in happier cases no fatal conditions arise. It is then that we see an amount of restorative power as surprising as the terribly rapid, mortal issue in the grave examples is startling. New bone forms from the periosteum around the old, like the bark of a tree around the old wood. The top and bottom of this new case, or capsule of bone, are united with the epiphyses, and when the piece of old bone, or *sequestrum*, is removed, this capsule of new bone gradually contracts to the dimensions of the old, though seldom quite so far, and then assumes the proper figure of the bone, having a medullary canal within it as before. The history of these milder cases, however, here merges in the general history of necrosis, which we shall presently describe.

Osteo-myelitis.—In speaking of periosteal abscess, we mentioned the frequent co-existence of inflammation of the medulla of the bone with the periostitis in that disease. Indeed, the cases are rare in which the periosteum or medulla alone is inflamed. Yet though this is true, we find it convenient to recognise as distinct classes those cases in which the periosteum on the one hand, or the medulla on the other, is the chief seat of the disease. Using the term osteo-myelitis as a general name for all cases of inflammation affecting the marrow canal and the lining of the cancellous tissue, the application of it becomes very wide, and includes affections of many degrees of severity and of several distinct varieties of disposition. Thus, in the neighbourhood of diseased joints the cancellous bone has its medulla nearly always, if not quite always, in a state of inflammation, the result of which inflammation differs according to the disease in the joint. For instance, in chronic rheumatic arthritis the inflammation is generally plastic, and leads to induration of and outgrowths from the bone. The same is the result in chronic traumatic inflammation; but in scrofulous disease of the joint the bone is generally very thin, the effect of the inflammation being an absorption of the *tela ossea* until what is left is often very fragile and greasy, becoming pellucid and bending under pressure of one's fingers. These forms of osteo-myelitis, however, come more properly under the history of joint diseases. Besides these instances of osteo-myelitis, which may be properly regarded as incidental to the course of other affections—though some think that many so-called strumous diseases of joints arise in this way—we meet with cases where the inflammation of the medullary tissue is undoubtedly the chief and primary disease. These cases fall into two groups: the first where the inflammation is diffused in the interior of the bone indefinitely, *diffused osteo-myelitis;* the second where the inflammation seizes upon a limited spot in the bone, *circumscribed osteo-myelitis.*

Diffused osteo-myelitis is found both as an *acute* and as a *chronic* disease. Acute diffused osteo-myelitis is a very important malady, and has received much attention since the part it plays in the production of pyæmia has been recognised. It is, indeed, the most frequent cause of pyæmia after amputations and resections, and also after severe blows on the head, such as inflame the cranium and its sinuses without fracture. When the bone thus affected is examined, the medullary cavity and cancellous substance are found to contain pus, which either fills these so as to form an abscess in the cavity, or is scattered in small collections about in the medullary tissue. Or else the pus is not evident; but the medullary tissue is spongy looking, red and tumid; it may then be very difficult to decide upon its state even by microscopic examination. For you know that the reddish marrow of cancellous tissue naturally contains more or less of a substance full of lymphoid cells not unlike pus-cells. These cells are often reddish, and not long ago Dr. Naumann advanced the hypothesis that blood-cells arise in the marrow, and that these red lymphoid cells are really blood-cells in process of development. Besides the ambiguity so arising, there is generally so much oil in the specimens that you are much impeded in your examination by fat-globules, and hence it requires a very careful inspection of all the characters of the case to come to a secure conclusion on the state of the bone. Sometimes there is lymph or pus in some parts, while in others the spongy reddening of the tissue prevails, the former parts being of a sulphur colour, the latter contrasting sharply by the brightness of their redness. We once met with a case of this acute diffused osteo-myelitis as the only secondary suppuration in a case of pyæmia from a carbuncle in the back, so that it may be the effect as well as the cause of pyæmia. When the inflammation is in this acute form the bone often dies throughout, and so may be found lying denuded of its periosteum.

But in some of the cases the inflammation is not diffused so freely, but limits itself to the parts of bone around the medullary canal. You see this frequently enough revealed in the form of a sequestrum from the interior of a bone after amputation. When the bone has been involved in acute inflammation of the stump and the suppuration has extended up the bone in the form of osteo-myelitis, it goes higher up the interior than the exterior of the bone, and the consequence is that the interior of the bone dies to a greater height than the exterior, and when the *exfoliation* of the dead portion is complete and the *sequestrum* separates there is a long piece withdrawn from the interior of the bone in continuity with the piece which lay exposed at the dead end of the bone. This extension of suppurative inflammation up the medullary cavity is always dangerous, tending, as we said, to produce pyæmia, though the frequent separation of these conical sequestra shows, that pyæmia is not a necessary result of such myelitis. Whether pyæmia shall occur or no in these cases is not altogether determined by the extent of the osteo-myelitis. Fatal pyæmia often is associated with a very little myelitis.

It has been proposed to remove the bone by a second amputation in cases of osteo-myelitis with signs of incipient pyæmia after amputation. The theoretical arguments are insufficient to decide in regard of this practice, but some published results of the practice by Prof. Fayrer and Mr. Holmes appear to bear importantly in its favour.

Chronic diffused osteo-myelitis.—Sometimes we find the whole of a bone enlarged, and its texture either greatly condensed and indurated, or with the cancellous tissue yet open and spongy, but full of lymph in various stages of disintegration or ossification, the indurated and lymph-charged parts prevailing more or less in different instances. We have seen specimens of the femur diseased in this way, when there was no part actually dead. The femur appears to be more liable than other bones to this affection. In some cases we meet with the same condition in a rather more acute state, the shaft of the bone being red and soft, and the soft cancellous bone being infiltrated with lymph more or less resembling pus. In all these cases the surface of the bone and periosteum share in the disease. The course of this affection is, however, very different from the acute osteo-myelitis. The cases generally are of several years' duration. The soft parts about these bones are greatly altered, the periosteum united with the muscles, and these largely changed to fibrous tissue. Some examples may have been syphilitic, but it has not been possible to prove such a cause in others of them. A lower degree of ostitis is sometimes met with, especially in the cranium and especially as the result of injuries. Thus, you may see the whole calvaria covered with a number of bony granulations, while it is thick, dense, and has lost its diplöe. The soft parts, within and without, partake in the inflammatory irritation, which thus may deserve the name cephalitis. The same is true of the spine; signs of disease respectively similar are not uncommon after severe injuries to the spine, but the anatomical effects are not so well known; we have found the vertebræ thick and rough in such cases. The history of injury is often remote, so that the suspicion of a constitutional cause, such as syphilis or rheumatism, cannot be averted.

Circumscribed osteo-myelitis.—This also occurs both as an acute and as a chronic disease, though the distinction is less significant here, as the cases which arise acutely generally run a prolonged course, and so come ultimately to much the same character as those which commence gradually. Acute circumscribed osteo-myelitis is a rare affection, many well-described cases of which, however, are to be met with. The disease produces intense local pain, and after a period of great suffering, aggravated in nightly exacerbations, pus is found to have formed, being discovered either by the surgeon's exploration or by the spontaneous appearance of a swelling. Circumscribed chronic osteo-myelitis differs only in beginning more slowly and insidiously. Some of the cases recover after evacuation of pus from the bone by drilling or trephining; when the course is adverse, and the diseased parts come under anatomical observation, the case has generally lasted a long time, and the limb affected has been removed after many patient endeavours to obtain a cure. We then find the diseased part, which is generally the articular end of a long bone, thickened, swollen, reddened, and a little softened; its cancelli full of lymph or pus, or else hardened and condensed by the ossification of the new product within its cancelli. This state rarely proves sufficient to render the limb hopeless, unless there be also present either a *chronic abscess* in the enlarged and inflamed bone, or else necrotic fragments lodged within the bone. This is thickened and diseased at the spot, with one or more sinuses, leading, perhaps, into a large joint, as well as to the surface of the limb. Perforating sinuses may be found without necrosis, as in an interesting case by Mr. Holmes, who believes that some sinuses may form by deep ulcers extending from the articular surface of the bone. The periosteum is often a great deal involved, so that it strips readily, or is already separated, and under it, or more frequently on the articular surface, a view of the bone may disclose eroded patches of ulceration, forming *simple ulcer* of bone, or *caries simplex* as it is sometimes called. This must be distinguished from true or fungous caries by the comparative firmness of the bone upon which the simple ulcers occur, a thin layer of condensed bone being beneath the ulcerous surface, and by the absence of the characteristic more or less widely extended caseous infiltration of true caries. We, however, find the limits that distinguish ulceration and caries of bone naturally vague, and in some cases it is very much a matter of choice which view we take of the nature of the disease. Indeed, as we shall presently see, caries is nothing else than a cachectic ulcerative osteo-myelitis, so that its relation to other obstinate forms of chronic ulcerative osteo-myelitis which we are now describing will naturally be very close in many cases.

When the small bones of the wrist and tarsus are affected with chronic inflammation, the disease has a course very similar to that of osteo-myelitis of the ends of the long bones. But there is often even a greater difficulty in distinguishing such simple chronic inflammation from caries, especially as the mechanical attrition of the ulcerous surfaces of the articular ends may wear away the bone, and waste it into a very deformed shape, yet there is no doubt that many cases of chronic tarsal and carpal disease are simply inflammatory, their obstinacy being due to the death of the inflamed portion of the bone. Necrosis of bone is frequently caused by the inflammation becoming acute, and then we often find, on examining the diseased parts, that the bone, where not necrosed, is in a healthy condition, and covered with strong granulations, or, perhaps, simply indurated by partial ossification of new products within it. In such cases the removal of the dead bone, when it is possible, forthwith alters the whole course of the malady, and repair at once commences steadily, progressing to entire cure, with or without reproduction of the bone, according as the periosteum has or has not escaped destruction. On the other hand, a small patch of caries has often been known to delay or wholly prevent the hoped-for cure after successful removal of dead tarsal bones or resection of the articular ends of long bones.

The purest examples of simple chronic osteo-myelitis are those that result from severe injuries, such as gunshot wounds or compound fractures. Many cases have occurred wherein, long after the healing of injuries of these kinds, inflammation has again appeared in the bone, leading to the death of a part of it. Sometimes minor degrees of injury, such as occur in children from blows on the head or falling from the erect posture, have produced ostitis of the cranium, with necrosis and separation of considerable portions of the calvaria. We have before described the more diffused effects of such injuries.

Abscess within a bone, at least in the form of a considerable collection of pus without necrosis, is rare; it occurs generally in the cancellous tissue of the long bones, especially of the femur and tibia. The bone is in typical cases expanded by an extension of the suppuration in the internal tissue, with a simultaneous formation of new bone under the periosteum, as the latter is pushed out by the increasing abscess. In this way the end of the bone may enlarge to a great size, and such was probably the mode of origin of some of those large cavities in the ends of dried bones which are to be found in museums under the old and absurd name of *spina ventosa*.

Ostitis.—We have described periostitis and osteo-myelitis, so far, as separate affections, but in most cases the inflammation implicates both the periosteal surface and the medullary tissue at the same time. The result then is simply a sum of the effects of periostitis and osteomyelitis. We have frequently alluded to this combination of superficial and deep inflammation, but it requires to be specially mentioned that the name *ostitis* is given to such an implication of the whole substance of the bone in inflammation.

Syphilitic inflammation of bone.—Syphilis produces periostitis, osteomyelitis, ostitis, caries, and necrosis, in short every one of the kinds of inflammatory affection to which the bones are liable. In this the syphilitic bone affections resemble the syphilitic diseases of the skin, which you know are equally manifold in their forms, exhibiting all the simple kinds of cutaneous disease modified through certain peculiarities of colour, &c. The effects of syphilis on the bones are often characteristic and recognisable. The leading character is that which marks all tertiary syphilitic lesions, namely, the limited area of the diseased change, and its productive character. Circumscribed thickenings, and low elevations on the shafts or flat surfaces of bones, are the most common effects of syphilis. These are well known under the name of "nodes," but in some cases the whole bone or many bones, even to the whole of the skeleton, may be affected. The disease also assumes all degrees of severity, and extensive necrosis may occur, though fortunately it is not nearly so common now as formerly, when the treatment of syphilis was fatally active. Even now necrosis of the bone may be induced by unnecessary incisions into fluctuating nodes, and a curable disease thus may be made almost or quite incurable. When necrosis occurs through syphilitic inflammation, the bone often presents peculiarities by which the nature of the disease may be recognised. This happens through the fact that the disease generally progresses for some time in the affected spot before necrosis takes place. In the first stage of syphilitic ostitis, as usual in all ostitis, the bone is rarefied by increase of the proliferating medulla at the expense of the *tela ossea;* thus the cancelli are enlarged, and the superficial hard layer is rendered porous. This process goes on until the affected part becomes more or less spongy before the necrosis comes to pass. Besides this, the dead piece of bone generally has a rounded figure with indented edges. This porous, round, indented sequestrum is very different from the smooth sequestrum of common inflammation. Such necrosis is generally seen on the cranium, and there also we usually meet with another characteristic effect of the same process of absorption of bone in the form of depressed patches; the slow, circumscribed, rarefying ostitis here, instead of leading to the death of the bone, removes the tela ossea and softens the bone, so that the superficial layers of it sink in over the space affected, forming a circumscribed depression, marked with radiating vascular grooves. The depressions from the two tables of the skull may meet, perforating the bone completely. Under these circumstances the matter which fills up the open cancelli is not pus, but granulation tissue, so that no suppuration accompanies it, hence the name *caries sicca,* by which it is known. Another peculiar form of syphilitic bone disease, also generally found on the calvaria, consists in the erosion of the surface along curiously curved sinuous outlines, surrounding and partly enclosing areas, under the surface of which enclosed parts, the erosion extends so as to throw off a thin scale of the bone. Here, too, the substance that forms in the groove is granulation tissue. We have known it extend through the vascular openings in the skull, along the vessels, and reach the sinuses of the dura mater, causing remarkable thickening of these, and in one case entire occlusion of them. After the more active rarefying stage of syphilitic bone disease has passed off there is always a rapid and effective hypertrophic tendency in and around the affected part, so that syphilitic bones are always heavy and hard. In caries sicca and sinuous erosion the induration is generally not accompanied by periosteal elevations.

Caries.—We limit the term *true caries* to what is called *caries fungosa* by those authors who call simple ulcer of bone caries simplex. *Caries fungosa,* or true *caries,* is that disease of a bone which corresponds to scrofulous inflammation of the soft parts, and it shares all the obscurity of this class of diseases. By scrofula we mean the slow caseous inflammations that arise without sufficient extrinsic cause, and do not show that tendency to recovery which characterises inflammation from injuries in healthy subjects.

The word caries is in constant use, and yet there is, perhaps, no word in all the pathology which is of less certain application. Thus, Sir J. Paget gives it no special place in his work, describing the changes others call caries under the name of tubercle of bone, while the nomenclature issued by the Royal College of Physicians gives caries as one disease and scrofulous or tuberculous disease of the bone as another disease.

The best way to realise the question for settlement will be to ascertain what it is that is practically called caries by those who use that term. We find the state pretty well understood and agreed upon. The chief characters of the disease called true caries are these: —1. The bone is softer than natural, being rarefied by the transformation of its *tela ossea* to medulla, by decalcification and multiplication of the lacunar cells. 2. Its medullary tissue is charged with inflammatory matter disposed in patches in the expanded cancelli, the inflammatory matter having more or less of a caseous quality in the centres of the patches, and so resembling the usual products of scrofulous inflammation which are called "crude tubercle." 3. The bony substance is not only soft, but itself undergoes fatty degeneration of its cells, and breaks down, yielding more or fewer small fragments, crumbling away spontaneously by what has been called "molecular necrosis." 4. The bone has a bare surface either on the periosteal or articular face, or else towards the cavity of an abscess, or of a chasm containing dead bone. 5. This surface is irregular and eroded in its appearance, and shows more or less of weak, pale, flabby granulations, which may be large and tender, as in caries of the internal ear, when they may form polypoid projections into the meatus exquisitely sore. 6. The discharge, instead of being purulent, is more or less watery and sanious, perhaps containing blood. Its effect upon the soft parts it passes over is a greater irritation than that caused by "laudable pus." 7. The surrounding bone, instead of being thickened, as it always is in simple ulcerative disease of the bones, is healthy, or in other cases it is greasy, being filled with an unnatural amount of fat, an interstitial absorption of the bony texture progressing to such an extent as often to make the bone yield under the pressure of a finger. When the bone shows these characters it is said to be carious, and there is, we think, no doubt about the matter as far as this. But the difficulty comes when the question arises whether this state constitutes a peculiar and specific disease of the bone, some thinking that any unhealthy ulceration, due to syphilis, scrofula, or even general weakness from any cause, is equally caries, while others say that caries of bone is quite a peculiar condition, and may be met with in persons who exhibit no constitutional weakness at all.

You will find, however, that this kind of dispute is not at all peculiar to the question as to the nature of caries. It equally applies in exactly the same terms to all those other instances of chronic suppuration which arise from insufficient extrinsic cause, and tends little to heal, and which are called by the general and very useful name "scrofulous." In so-called "scrofulous" suppuration of the kidney it is equally true that you are not always able to show the described constitutional signs of scrofula. We think it is better to abandon these described signs of scrofula altogether, for their range and application are hopelessly inexact, and to limit the meaning of the term scrofula to a state of tissue that reveals abnormal vulnerability and abnormal tenacity of injury, showing these characters by chronic suppurative destruction of the affected part without sufficient of the reparative formative process by which the suppurative inflammations of healthy persons are terminated and healed. The intense obstinacy of caries has favoured the disposition to ascribe to it a specific and peculiar nature. The impossibility of cure has been thought to be a specific distinction of true caries from common inflammation. When, however, we consider the conditions of bone in regard of the process by which ulcers heal, we can understand how imperfections in the formative activity in ulcers should in bone be of far greater consequence in impeding cure than in the case of the soft tissues. You know that in the healing of ordinary ulcers the parts are restored by two processes, the first of which is the production of new matter to fill the space, the second is the closing up of the parts around, either by their swelling or by the contraction of the new tissue formed in the other process. Now, of these two processes the second is often the more efficient, and is quite necessary if any considerable loss of tissue has occurred. We are, indeed, too familiar with the unfortunate results that arise when only the first process is available, and the second is prevented by the state of the parietes of the ulcerous space. Thus, it is to the rigid and unyielding walls of the cavities they occur in, that the obstinacy of phthisical vomicæ and of anal fistulæ is due, so that when a surgical operation enables the walls of an anal fistula to close in upon each other the healing of the sinus is soon achieved. If, now, we apply these reflections to the case of carious bone we see that the bony tissue is too rigid to allow of closure of the ulcer by collapsing around the loss of substance. No closure by swelling or collapsing of parts around can occur, and hence all the obstinacy of fistulous disease is to be expected, especially as the formative process is enfeebled and impeded by the resistance which the hard *tela ossea* offers to the free growth and nourishment of the granulations themselves. We have already said that in many cases of obstinate disease of the bones, leading ultimately to resection or amputation, the state of true caries is not present, the chronic inflammation of the bone not being accompanied by caseous softening, as in caries, but by thickening and induration. The obstinacy will then be found due to the existence of a sinus or abscess in the bone or a piece of necrosed bone imbedded in it, the inability to heal being due to the mechanical impediments to the closure of the cavity. As to the so-called syphilitic caries, we have always found that the so-called caries in syphilitic cases differs essentially from the true caries of chronic scrofulous inflammation in this, that the bone about the disease is thickened and not rarefied, as in the chronic scrofulous disease. Indeed, in so-called syphilitic caries of the skull a very great increase of weight of the whole skull is commonly present, whereas the whole of the dried femur in cases of true carious disease may not weigh more than two fifths or two thirds the normal weight of the bone. Caries usually affects the short bones of the wrist and tarsus, the vertebral column, or the ends of the long bones. The effects of it in these different parts are rendered different through the various anatomical relations and physiological circumstances of the bone affected. Thus, the small bones of the wrist or tarsus will undergo caries for a greater or less time, and so be reduced to four fifths, three fourths, or two thirds of their proper size, and then very often comes a new result, for the disease, extending all around the bone through its smallness, kills it by separating it from its periosteum and so from its vascular supply. For in carious inflammation, unlike healthy chronic inflammation, no new vessels are formed to connect parts separated by the lymph that has collected between them. Thus, *caries of the small bones* is after a while usually complicated with necrosis. The facts often come before the surgeon apparently in the reverse order in this way. He operates for the removal of a piece of dead bone from the wrist or ankle of a small child; afterwards the case does badly and amputation is required, when it is found that the necrosis was only a part of the disease, the natural result of a caries which had spread so as to involve the entire surface of the bone or so much of it that the whole bone had perished, the caries afterwards spreading amongst the other bones, and going on, perhaps, towards destroying others in the same way as that which had been removed. *Caries of long bones*, on the other hand, can implicate only a relatively small extent of surface, and hence is oftener uncomplicated by necrosis, since, as the disease extends slowly around the bone, the supply from within the medullary canal has time to enlarge itself and meet the new demand. Caries very rarely affects the shafts of the long bones; it is usually limited to the epiphyses. When caries attacks the bones of the extremities the products of inflammation may be long retained or even reabsorbed, being in small quantity, and consisting only of granulation tissue without pus. But when pus is freely formed the abscess is readily opened, and the pus is then found to be watery with flocculi of caseous matter suspended in it. Analysis shows such pus to contain excess of lime salts, and often small fragments of bone may be detected in it. But it is to be noted that all the abscesses and sinuses around carious bones do not always reach the bone or joint. Some abscesses arise by local suppuration in the inflamed tissues outside the joints, forming circumscribed abscesses, which, being opened, form sinuses.

Caries of the spine offers a new condition from the pressure of the weight above the carious part, which necessarily tends to bend the spinal column, and as the bodies are the parts of the vertebræ affected, while the spinous processes, &c., generally escape, the bend takes a forward direction, an angle with backward projection being produced by the sinking of the bodies of the vertebræ above to fill the hollow caused by the wasting and removal of the bodies at the diseased spot. This angular curvature is called *Kyphosis*.

The disease in the spine is also peculiar through the presence of the intervertebral substance. It is a curious fact that disease of the spine commonly shows itself first in the intervertebral substance, a fact insisted on by Mr. William Adams and fully borne out in our experience. It may not yet be certain whether these cases should be regarded as a special kind of disease, distinct from the true scrofulous caries, which, according to some authorities, always begins in the bones. Certainly we have frequently met with cases where the intervertebral substances were specially affected, yet the bone was decidedly carious. And then it was clear that the disease in the bone had been propagated from the intervertebral substance; for instance, you may often find a vertebra apparently destroyed in its middle by disease, but on closer examination the two portions which appear to be the remains of one are found to belong to two separate bones, the disease having commenced between them. This has happened so frequently that we cannot agree in Rindfleisch's statement that in caries of the spine the intervertebral substance is either not at all implicated or only late in the course of the disease. But it is probable that in many cases of so-called caries of the spine beginning thus the disease is not scrofulous. Indeed, the great mobility of the spine, and the pressure to which its parts are subjected, explain the unhealing quality of many neglected inflammations due originally to a strain or blow, without our being obliged in explanation to have recourse to any peculiarity of constitution on the part of the patient. But we think the character of the morbid process is very different in the two classes of cases, of which one is typified by the spinal disease in weakly scrofulous children, and the other by the spinal disease of otherwise healthy adults. In the scrofulous cases the bones are soft and little or no sign of repair is present, the conditions being just such as we have described as characteristic of caries. On the other hand, in cases which occur from injury in healthier subjects the bones are indurated and new bone is formed around the seat of disease, and the tendency to repair may be so effective as to end in complete ankylosis of the bones in their new angular position, causing the hunch-backed figures we not unfrequently meet with. Such recovery is, indeed, favoured by the angular curvature bringing the bones together within reach of each other. All stages of the destructive and reparative processes in bones may be seen on our shelves. In this example, which was taken from the body of a strong and previously healthy man, there was not the slightest indication of scrofula, and, according to the history, no doubt the disease began in the intervertebral substance. The man strained his back in carrying a weight, the intervertebral substance was slightly lacerated, some lymph was thrown out, and subsequently a gradual destruction of the whole cartilage took place, and at last the adjacent surfaces of the bones were involved, as seen here. You may see, then, in some of these specimens a yellow soft matter, affecting only the intervertebral substance; in others, a destruction of this substance with the adjacent bones; and in others, numerous vertebræ altogether destroyed. You will see also in these specimens how repair takes place. In some instances the fragments of bone are held together by ligamentous tissue, and in others they are firmly conglomerated into one irregular mass of bone. An extreme angular curvature necessarily results from the vertebræ thus falling together. You will observe how remarkably the medulla follows the curvature of the bone, and how it escapes mischief in numerous cases. *Lumbar* and *psoas abscess* are shown very imperfectly in these dried preparations. They usually result from caries of spine. In this case, where there is disease between the fifth and sixth dorsal vertebræ, a lumbar abscess existed on one side and a psoas on the other. Sometimes the articular processes are affected, or the adjoining ribs are involved in the caries.

Disease of the *odontoid process* may occur as a separate affection. In this specimen you will see a remarkable instance of this process becoming carious and making its way out through the pharynx, the patient completely recovering.

Disease between the first two vertebræ, the atlas and the occiput, is not uncommon, and results in ankylosis, numerous specimens of which you will see on our shelves. In all our specimens of disease of the *cervical vertebræ* you will see that the abscesses which accompanied them opened into the pharynx.

Caries of the cranium we have already alluded to, but here are other specimens which show rather a simple erosion as effects of lupus or facial cancer; you will perceive there is no new deposit of bone around the decaying edges, as in caries, but a simple erosion of the structure.

Necrosis.—We have frequently had occasion to mention necrosis or the death of bone as a consequence of severe inflammation of its medulla or periosteum. This is the most common cause of necrosis, but it is met with also as a result of accidents. Fragments may be entirely separated from their connections, and hence may perish; more frequently the periosteum is stripped off the bone, and perhaps injured or destroyed at the same time, or the bone is exposed to the air through the wound in compound fracture. Under either of these circumstances there may be any degree of inflammatory complication co-operating with the injury to destroy more or less of the bone. Thus, a simple inflammatory action may be set up and a speedy cure result; or the bone may become ulcerated in the same way as the soft parts, and after a more lengthened period form granulations and heal; or, again, if a large portion of the periosteum be destroyed, the bone is likely to die, for the periosteum being removed, the vascular supply is cut off and the exposed surface mortifies, the bone becoming white and subsequently black. To a certain depth only does this change occur, for the deeper seated portion of shaft being well supplied by the medullary membrane and adjacent vessels, a line of separation ensues between the part thus nourished and the exterior.

Inflammatory granulation tissue forms along this line by decalcification of the bone in it and production of the granulations from the soft substance thus set free, so that in the course of some weeks the dead portion becomes detached, leaving the living raw surface below covered with vascular granulations; these then become changed partly into new bone and partly into a new periosteum, which forms, with some fibrous tissue and the adjacent soft parts, a cicatrix. A small scale of bone may thus come off, or, if some inches of the shaft be exposed, a portion extending to the medulla may come away; if more, of course there is danger of death to the whole bone. This process of separation of a portion of dead bone is called *exfoliation;* it may be well seen in these specimens of diseased craniums, where, owing to the distinct vascular supply of the interior and exterior of the skull, large portions of the external table have come away without interfering with the deeper portions; you will here see a large piece of bone, the size of the palm of the hand, in the process of separation, and on looking at it you will also see how it is bevelled off so that the external surface is much larger than the internal. Even in this specimen, where necrosis has resulted from severe injury, involving the whole thickness of the skull, the internal table is only brought away from a single spot in the centre of this large piece, which has nearly all come from the external table.

We so far have spoken of the dead bone exfoliating and coming away as if this were a matter easily accomplished. But, unfortunately, this is far from being the case; indeed, it is the great difficulty that attends removal of the dead bone which has drawn so much attention and brought so many long names to the description of necrosis and of the parts concerned in it. When the piece of bone is separated or *exfoliated* it is called a *sequestrum.* This sequestrum generally does not lie free among soft parts, it is more or less enclosed in bone. This enclosing bone may be either the surrounding parts of the original bone, as when the portion which died was deep seated within the natural bone, or else, if the necrosis was superficial, it will be new bone, which arises in the following way. The periosteum, whose separation is the cause of the death of the bone, is thrust in the separation to some distance from the bone, being separated from it by blood or pus, according as the disease was due to injury or inflammation. The deeper layer of periosteum which immediately encloses the bone is softer than the rest of the periosteum; it has well been called the *cambium* layer, since, like the cambium in a tree, it is the seat of the active growth; it also has a soft consistence, so that it tears readily. This cambium layer has a very great ossifying power, and when displaced from around the bone it begins to form bone in its new situation as soon as destructive inflammation subsides. Thus, it creates a case of bone around the dead part of the shaft. This case of new bone is called the *capsule.* The new formation of bone always extends some distance beyond the necrosed part of the shaft; it goes on under the periosteum of the remaining part of the old bone at both ends, uniting there with the old bone. Thus, the new periosteal capsule of bone is made continuous with the remaining parts of the original bone. It is not quite a complete sheath, but shows a few round openings, which lead through it inwards to the space between it and the sequestrum and outwards into those fistulous channels which conduct the discharge to the surface. When such a bone is dried these openings, called *cloacæ*, are found as round orifices in the capsule of bone, with everted, rather prominent edges. Surgeons are in the habit of passing probes through these cloacæ and feeling for loose bone within, which, if found, is removed if that be practicable. We may here mention to you that, in a specimen like this tibia, where the new bone is bent, it is probable that the attempt to remove the old bone was made too early, for it is necessary that it should remain a sufficient length of time as a model for the new bone to be formed upon; if not, the latter, being soft, is acted on by the muscles and becomes distorted; the same happens in the lower jaw if necrotic bone be removed at too early a period. You will learn in the surgical lectures at what period interference is necessary; but we show you these specimens that you may understand how the new bone is modelled upon the old. In young persons necrosis of long bones affects the shaft alone, and, the epiphyses remaining, the joint escapes; but this is not universally the case, and in adults the disease is very likely to continue to the extreme ends of the bones; in such instances, with the joint involved there can be little hope of repair, but we think it possible for an epiphysis to be renewed, although with ankylosis of the joint, as seen in the specimen.

There are some special circumstances under which necrosis occurs which require to be known. Those engaged in the manufacture of lucifer-matches suffer necrosis of the jaw-bones. The mischief is set up through the action of the fumes of phosphorus; it is called in the match trade "the match-maker's disease." It commences along the alveolar border, perhaps in the seat of decayed teeth. The disease spreads slowly, and is usually accompanied by a free formation of spongy new bone on the margin of the necrotic portion.

Another remarkable special instance of necrosis is in the case of some children after exanthems, a portion of the alveolus of the jaw-bone perishes and separates, bearing with it the milk tooth and sac of the permanent tooth; no more of the jaw comes away than is required to liberate the teeth.

Injury.—The bones pay a tribute for their firmness in their liability to fracture. In children and young persons the fracture may pass through the epiphysial cartilage, so that separation of epiphysis results. The bones may be perforated or crushed by gunshot, or cut with sabres, &c. Sometimes a mere bruising of bone, especially of the cranium, when little is feared from it, may set up suppurative inflammation, leading to acute necrosis, pyæmia, and death.

Fracture.—When the broken bone is exposed in a wound leading to the surface the fracture is called *compound*, otherwise when there is no wound exposing the bone the fracture is called *simple*. This distinction corresponds to a very important difference, since the healing of simple fracture is a very simple process, unattended with suppuration or serious inflammation, while the healing of compound fracture is a complex process, much slower, attended with suppuration and granulation, and involving commonly no little danger of osteo-myelitis, with its terrible follower pyæmia.

In the case of simple fracture a soft matter forms around the ends of the bones and ossifies, thus uniting them together. When the ends override each other, or are not kept steady, a large quantity of this ossifying material is rapidly produced, so as to form a large mass around the fracture; but if the apposition of the ends be even and close, the amount formed is small, so that the swelling may not be detectable. Whence comes this ossifying matter? Whenever a bone is broken there is some bleeding from the bone itself, but more especially from vessels torn in the sharp jerk that accompanies the breaking of the bone. It was formerly a much contested question whether the blood thus poured out undergoes ossification, so as to be itself the bond of union, but this is definitely settled in the negative.

The blood is regarded as so much foreign substance, which is only in the way, and has to be removed before or during the reunion. It is now known, from numerous careful observations, that the new ossifying substance is formed from the periosteum principally, but partly also from the medulla of the bone. If the bones be not well placed together, other tissues, where they are continuous with the periosteum, will take to forming the bony union, as though they were infected by its kind of activity. Whether the periosteum or the medulla produces the greater proportion in any case depends on the circumstances of the case. If the ends be left more or less overriding, so as to meet by their sides, then the periosteum becomes quickly much swollen and softened for two or three inches on each fragment; how much of this swelling occurs depends on how bad the position of the ends, and how unsteady their maintenance. The thickening is greatest at the end, and gradually diminishes up the shaft. The swollen periosteum of the fragments unites where they meet, and then a soft mass is found joining the broken ends. This mass is continuous especially with the deep layer of periosteum, and it has essentially the same microscopic structure, but is more translucent, being in some parts very much like cartilage from the hyaline appearance of the intercellular substance, and the encapsulated look of the cells. Indeed, it sometimes is so like cartilage that the name cannot be denied to it, but at other times it is more fibrous. This mass is called the "callus" or the *external callus*, as distinguished from the similar mass produced from the medullary tissue which closes the hollow broken end of the shaft, and is called the *internal callus*. It is also called *provisional callus*, because none of it ultimately remains unless it is wanted to secure continuity in the line of the shaft. The production of this swollen mass is exactly that process which we have described as "ossifying periostitis." Bone soon begins to appear in it in the form of spicules so arranged as to compose a sponge-like porous mass, in the channels of which are new vessels. This soft bony sponge commences about the third week, and during the next three or four months undergoes a modelling process, so that so much of it as is required to complete the shaft passes on to the formation of hard bone; this is done by the channels of its spongy tissue being filled up with concentric laminæ of bone, which narrow them to the proportions of Haversian canals, thus making hard bone; but the greater part of it is not needed for the continuity of the shaft, and it wastes away and is absorbed; so, too, any projecting ends of the fragments lose their prominence, and are rounded off until at last there is left no more than will complete the fragments into a column.

Meantime the fractured faces, on which of course the medulla is exposed, are undergoing corresponding changes more slowly and less largely. If the position be very good, little external callus appears; it may even not be detectible in a fractured tibia by examination with the finger; but the broken ends lying adjacent take on that form of action called *rarifying ostitis*, the lime salts disappear, and the cellular medullary tissue set free forms a spongy mass of callus that closes the end of the shaft, and meets with the corresponding mass in the other fragment. These masses unite with each other, so that the continuity can be at once restored by the ossification of this *internal callus*. Afterwards, when this callus has become thoroughly developed into ivory bone, a modelling process of atrophy reabsorbs that portion which filled up the narrow canal, so that in the end there may be scarcely a trace of it, and no sufficient evidence that a fracture ever existed. Thus the callus serves to maintain the ends in temporary union, performing the service of splints, and usually signifying the want or insufficiency of these. For provisional callus, as we have seen, is not necessary at all, it occurs mostly in those cases where perfect rest cannot be commanded. It is well seen in fractures of the bones of lower animals, made in experiments, from which, indeed, the description of the union of fracture has been too trustfully derived, leading to a too great prominence of the provisional callus in the history. For a visit to the accident ward will inform you that a fracture may at once unite without any provisional callus; you will not fail to find some cases of fracture of the tibia where the line of union can be scarcely felt. On the other hand, if the part be not kept pretty steady during the few months required for the modelling of the bone into its proper form, the provisional callus will not disappear, but will continue in the form of a permanent node of hard bone. This is well seen in our skeleton of the hippopotamus. That must have been a very unfortunate, or, perhaps, a very reckless animal, for he has broken all his ribs, without favour to any, except that others are broken in several places. At every injured spot there is a large hard node; no doubt the want of rest, and the necessary movement of the ribs in breathing caused the excess of bone formation. It is not very uncommon to find such an excess even uniting two or three ribs together if the injury has been severe. In this preparation of a fractured clavicle you see the large, spongy, external, temporary callus uniting the bones together. This spongy bone would have been absorbed when the ends of the bone were firmly united, but not so the hard nodes of the hippopotamus' ribs; they would be permanent.

When the fracture is *compound*, suppuration usually occurs about the broken ends, and the healing of the bone is effected by granulations forming and ossifying, as in ulceration of the bones.

False joint.—When the fractured ends of a bone do not unite a false joint is formed. You must remember, however, that it is quite the exception to have a new perfect joint produced; all you find is that the bones, which either are too far separated or kept apart by other tissues, or are deficient in repairing power, form, instead of bone, only a ligamentous tissue, which allows free play between them. Thus, in this specimen of a humerus, where a fracture had failed to unite eight years before, the ends, as you see, have become pointed and firmly joined by a strong ligamentous band, but there are no structures such as would constitute a true joint. Probably where there is a broader surface of contact more of a joint is produced. Thus, in this specimen of forearm, the ends of the bones are surrounded by something like a capsular ligament, and within is a synovial sac and fluid.

Some seats or kinds of fracture have special points in their history which we must draw your attention to. Thus, *fracture of the neck of the thighbone* is far from infrequent, occurring usually in an elderly person who has suffered a sharp fall, and is then found to be powerless in one leg. We recently had before us an example of this fracture on both sides in a man seventy-two years old who had been knocked down by a cab. The case was deceptively like paraplegia. Much interest attends this form of fracture because it is very rarely, if ever, repaired by bone, and many reasons have been given for this. One is that all fractures in joints are seldom thus repaired, both because the breach is filled merely by a synovial fluid, so that there is an absence of the soft textures which would assist in supplying the necessary pabulum. But the chief reason is the difficulty of keeping the parts of a joint in perfect apposition: this reason obtains in other joints, for in the olecranon osseous union is rare, and as regards the patella its bearing is particularly evident, for in vertical fracture of that bone, where there is no difficulty in preserving contact, an osseous union usually occurs, while its transverse fractures never unite by bone. Another reason for non-union in the particular case of the cervix femoris is that the nutritious artery of the neck is divided, so that the only supply of blood to the head of the bone is by the round ligament. Old age, too, the time in which this accident occurs, has been considered instrumental in the prevention of union; but of this there is great doubt, unless it be owing to the degenerative changes which might be previously going on in the neck of the bone, for union in other parts of the skeleton may take place in old people; and, on the other hand, a want of osseous union in the joint is observed in the young. For example, this bone is from a child who received a severe injury, and broke the upper part of the os femoris both within the capsule and without; the external fracture appears to have been repaired, but the inner shows no sign of cure. You will see, by observing these specimens of fractured cervix femoris, how little repair has advanced after several weeks or months, and if the parts have in any way united, it is only by some ligamentous tissue. Such a union is all that you can generally expect. In this specimen, which we obtained the other day from a woman who fractured the neck of the bone five years before, there is good ligamentous union, and, besides this, some nodules of cartilage between the broken extremities. In all these cases you will see how the faces of the fracture have suffered absorption, and the neck is thus considerably reduced in length. Amongst these specimens there are some which are labelled as doubtful instances of bony union; and the difficulty of giving a correct judgment is often very great, for this reason, that a change very similar to that which an injury might produce is caused by senile disease. An old person, for example, has been subject for a year or two to the changes, which we have shown you are so often present in the neck of the thigh-bone, and which produce a shortening of this part. If now, in consequence of such impairment of the joint, the person should fall, and owing to the bruising take to his bed, and a medical opinion be sought, the leg being found powerless and shortened, it might be thought that a fracture had occurred, and then, if the case be watched, and after a year or two a *post-mortem* examination take place, it is highly probable that the distorted appearance of the neck of the bone may somewhat resemble a united fracture; for if a section be made, the irregular form of the cancellous structure may display some fibres of bone corresponding in place to a supposed line of fracture; and if, moreover, the joint has been affected by chronic rheumatic arthritis, some new bone may have been formed in the surrounding parts. That form of fracture external to the capsule, in which the neck is driven into the broken trochanter, unites easily by new bone, which binds all the parts together. This injury is known as *impacted fracture.*

Fracture of skull.—This subject will be given in full in the surgical lectures, and we will therefore merely ask you to carefully regard these specimens; you will see that they can be divided into two classes of cases, which correspond with a similar practical division you meet with in the wards, according to the nature of the injury and the chances of cure. The distinction is simply this, some are circumscribed, and some diffused, and the difference arises in this way, that in the one class of cases the injury is due to a sharp blow struck with a light weapon, and in the other to a heavy blow from a larger body moving more slowly. In the first kind of fractures the violence does not extend its area much beyond the spot immediately struck. In the other class of cases the injury is diffused over the whole skull and its contents. The difference between the two kinds of cases may be well illustrated by what you have doubtless heard of in discussions between the two schools of naval artillery. There are, you know, two systems in one of which the object is to throw a small shot with high velocity, and so pierce the side of the vessel—this is known as the punching system; while, in the other, a large shot is thrown with low velocity, so as to strike heavily, and shake and rattle the timbers of the ship—this has been called the racking system. In just the same way, when a sharp blow is struck on the skull with a hammer or sabre, or with a bullet, the parts immediately touched alone receive the damage. In these cases, the injury being circumscribed, there is a fair chance of recovery by judicious treatment. It is to this class of cases that most of our numerous specimens of repaired fracture belong. But when a man falls from a height upon his head, or is struck by a falling beam, or a cart wheel, in short, when the momentum of the blow is due rather to mass than to velocity, the fracture then extends far beyond the point of contact of the injurious agent. It is interesting to trace out the conditions that then arise; what happens is generally this, that a fissure starts from the point struck, and runs towards the base of the skull pretty straightly, but with more or less comminution, according to the severity of the blow. The fissure usually runs through the temporal bone, making its way in front or behind the rocky part of the petrous bone, so as to pass into the middle or posterior fossa of the basis cranii. The reason of this is that the petrous bones, from their position, being hard thick masses nearly over the condyles, convey the force of impact from the spine, and throw too great a share of this force upon the parts with which they are continuous, so as to strain and break those parts. It is easy to see that, when the skull is compressed to bursting between the spine and the ground, there will be a leading tendency for the rent to occur in a line from the spine to the ground, *i. e.* from the base of the skull towards the vertex. If the fall is upon the front or back of the vertex, this leading tendency to burst along the direct line between the two points of pressure will induce the fissure to run down the frontal or occipital bone into the anterior or posterior fossa; on the other hand, if the fall be immediately on the vertex, the momentum of the body, acting through the spine, will drive in a rim of bone around the foramen magnum, as you here see.

When the fracture leads into the middle ear, and ruptures the tympanic membrane, or runs into the pharynx, then there is bleeding into the ear or throat, as well as frequently an escape of the cerebro-spinal fluid or even of the brain tissues. The fracture is apt, in passing in front of the petrous bone, to tear through the line of the Eustachian tube, which is a weak line; or, in passing behind the petrous, it is apt to crack through the very slight partition between the jugular fossa and the tympanum; or, not unfrequently, the petro-occipital suture is partly forced, and the spongy end of the petrous broken across into the Eustachian tube. In either of these ways a road through the bone to the torn tympanic membrane is opened up; now, such violence is always accompanied by laceration of the dura mater and arachnoid. If the naked eye does not discern the rent, a lens will show the surface of the dura mater to be cracked in severe injuries of this kind. The visceral arachnoid is nearly sure to be also broken, and thus blood and cerebro-spinal fluid come out *into*, and then out *from*, the arachnoid space through the external ear. Or the fissure, passing through the roof of the pharynx, tears also perhaps the lamina cinerea, forming the floor of the third ventricle, so opening a route for the fluid down from the ventricles, through the bones, into the pharynx. In some remarkable cases, collected by Mr. Prescott Hewett, a fracture of the calvaria communicated with the lateral ventricle, and so the ventricular fluid came away directly through the wound. Again, we have shown it to be nearly certain that in the common fracture about the jugular foramen the pneumogastric nerve is dragged on enough to tear the very delicate wall of the lateral horn of the fourth ventricle, with which it is closely united; so that in these fractures the fluid that comes through the ear is directly derived from the fourth ventricle. It was Mr. Hilton who first in this country proved that the fluid that escapes from the ear in these cases is of the same nature as the cerebro-spinal fluid.

Another point of interest in fractures of the skull is the so-called *contre coup*, a name signifying the tendency of injuries to show themselves at a point opposite the part actually struck. So far as we have observed, you never see the skull broken at the opposite point; the fracture of the bone is always where the blow was received, and extends from that spot as already described. So, too, the bleeding outside the *dura mater* is always at the point struck; in fact, this bleeding is from the meningeal vessels torn in the separation of the bone. This is, practically, a very important point; the blood is found in the form of a hard clot between the dura mater and the bone. But, on the other hand, the injury to the brain differs in its seat according as the fracture is of the kind we have called circumscribed or diffused. If the fracture is circumscribed the brain is injured at the seat of the fracture; and if it be exposed a hernia cerebri will occur, generally with an abscess behind it; but if the fracture is of the diffused kind, then the injury to the brain is always at the parts opposite to the point struck; for instance, if the fall is on the left side of the back of the head, the brain is torn in front on the right side. The part of the brain then injured is generally the prominence of the middle lobe and also of the anterior lobe.

It is necessary to notice how unequal are the circumstances of different parts of the cranium with respect to the liability to fracture. Thus the condyle of the lower jaw may be thrust up and break the glenoid cavity, or even be thrust through it; or the nasal bones, in a fall on the nose, may conduct the shock and thrust the parts of the ethmoid with which they articulate up into the cranial cavity; or the excessively thin roof of the orbits may be burst at a great distance from the blow: we have seen a piece of the size and shape of a shilling broken out of the roof of each orbit by a fall on the occiput. This was the only approach to *contre coup* of the bones we have met with;

or these thin orbital plates may be thrust through with a tobacco pipe or umbrella, when little notice has been taken of the wound through the eyelid. On the other hand, the frontal sinuses may be broken only in their superficial wall, and in the same way, through the presence of the diploë in other parts, the outer table only may be broken, the inner escaping. But more often a blow on the outer table breaks the inner table to a greater extent than the outer; this is because the vibrations are dispersed, radiating away from the spot struck, so as to widen the area of the force as it goes from the outer to the inner table. In rare instances the outer table is not broken, or scarce broken, and the inner table is considerably fractured. In children the skull may be bulged in to a considerable extent in consequence of a fall, producing a curious deformity. Sometimes the fracture reaches a suture and forces it apart, so producing what is termed *diastasis* of the suture. If this occurs in the sagittal suture the longitudinal sinus may be torn. In this specimen, which we recently met with, a Wormian bone in the angle of the lambdoid suture has been dislocated, and has turned half round, so that its pointed end stuck into the longitudinal sinus, causing fatal hæmorrhage. Indeed, if we were to mention to you all the curiosities of fracture of the skull, we should not be able to bring this lecture to an end. When circumscribed fractures are severe, and even comminuted, portions of the bone may be removed and yet recovery take place; and even in bad cases of diffused fracture, when the fissure reaches the base of the skull, recovery may result, though this is much more rare, as we have already said.

Here are several portions of skull, picked up from the field of battle, showing very severe injuries, which have been repaired, and effectually repaired, as proved by the subjects of them having been able to engage in service again. These long fissures have, no doubt, been caused by sabre wounds, and, though the inner table is reached, recovery has taken place. In this, where the bone has been comminuted, but portions not removed, a union has occurred between the pieces. Where there is actual loss of bone, as from trephining, repair does not occur; but the dura mater on one side, and the scalp with the new cicatrix on the other, become united into a firm membrane for the protection of the brain. In this specimen, where there has been loss of bone, but whether from injury or disease is not very clear, the membrane taking its place is seen to have a few spiculæ of osseous matter shooting from the inner table, and a few isolated deposits in its centre.

Fracture of the spine.—In fracture of the spine the line of fracture takes a *direction* downwards and forwards, and generally implicates at least two vertebræ with the intervertebral substance between them. Sometimes the rent traverses the intervertebral substance only, or, more frequently, separates this from the bone, in which case the injury is called *dislocation* *of the spine*, and is distinguished from fracture. But the difference is of no importance, and, so far as we have seen, the more carefully the injured spine is examined, the more surely is some injury of the bone discovered. In the *cervical* region, however, the intervertebral substance may be torn through without breach of the bone, and perhaps the same may happen in the *lumbar* region also. In the *dorsal* region the interlocking of the processes of the vertebræ is so close that there cannot be a dislocation without fracture. It will easily be seen that no considerable displacement of the upper on the lower fragment could occur without serious danger to the contents of the spinal canal; and, indeed, in most fractures of the spine there is a considerable *displacement* in the moment of the injury, although the bones are restored almost to their natural position afterwards by the action of the muscles. Hence the *spinal cord* suffers more or less severe injury, even to complete severance. But if the fracture is below the first lumbar vertebra, so that the nerves forming the *cauda equina* are implicated, these more commonly escape by reason of their comparative toughness and mobility. Hence fractures of the lumbar vertebræ, even when accompanied by considerable displacement, may be recovered from with a permanent unevenness of the spinous processes. When recovery is not reached, but the patient dies after many months' illness, a large quantity of *new bone* may be found, forming an incomplete shell around the broken part of the spine.

Particular interest attaches to the fractures *above the third cervical* vertebra, which cause instant death through stoppage of respiration, and which are sometimes mistaken for concussion of the brain, the spine having been forgotten in the post-mortem examination. Cases of fracture of the cervical vertebræ *below the third* generally live for two or three days, and then die from accident connected with imperfect respiration, or disorder of innervation of the sympathetic attended with a very high temperature. But in fractures in the *dorsal region* the sufferers generally live for several weeks, and then die either through the results of paralysis of the bladder, leading to suppuration of the kidneys, or through bed-sores and exhaustion or pyæmia; cases that end thus, after lingering many months with long-standing bed-sores, may show lardaceous viscera complicating the other conditions.

But though simple fracture of the dorsal spine thus usually has a course of several weeks, yet in some cases the fracture is accompanied by fracture of the *sternum*; in other words, the skeleton is broken across the thorax. Such cases usually die more quickly. On the other hand, some remarkable cases are recorded of life prolonged for years after fracture of the cervical spine, especially one by Mr. Hilton, in which the patient lived fourteen years quite paralysed as to his extremities, and yet became so clever, by practising the use of his lips, that he could at last sketch very excellently. The *spinous processes* of the vertebræ are sometimes fractured without injury to the rest of the column.

Fracture of the pelvis is also a very perilous form of fracture. Its special interest and much of its danger arise from the relations of the *urethra* to the pelvic bones, which frequently cause the urethra to be ruptured in pelvic fracture. You know that the urethra pierces and is firmly fixed in the *triangular ligament*, and that this ligament is connected with the pubic arch very firmly, while the pubic arch is the weakest part of the pelvic bones. It hence arises that, when this arch is burst violently through—and pelvic fracture requires great violence—the triangular ligament is torn, and the tear implicates the urethra; for this soft mucous channel forms a large weak spot in the ligament that gives way with especial ease. But, besides this way in which the urethra suffers, it may be crushed against the pelvic arch in falling astride a beam. In some very severe fractures we have known the pubic bones to be even forced back upon the sacrum so as to cut the prostate almost off the bladder, the bones recovering their usual relations afterwards through spasm of the muscles.

Separation of epiphysis.—This kind of accident can only occur in the young. The cartilage between the epiphysis and shaft of a bone forms a weak line in which a fracture might be expected to run. If this be borne in mind and looked for it will be found to be a form of fracture not infrequent in children. Knowing that the growth of bone is effected by a continuous ossification of this epiphysial cartilage, it is natural to apprehend that the disruption of it will lead to a check in the growth of the bone to its full length; and some degree of shortening is ultimately found, though it is matter for surprise how nearly bones so injured do often come to perfect length and form.

We have already mentioned another peculiarity of fractures incidental to youth, in speaking of the bending-in of the cranium occasionally in young children. The same kind of accident will occur in their long bones, bending the bone instead of breaking it; of course, in such cases there must be a certain degree of interstitial crushing and disruption of texture. This condition has been called *Greenstick fracture.*

Spontaneous fracture.—This occurrence generally signifies a diseased state of the bone, and often is due to *cancer* in the medullary cavity—a circumstance which will be indicated by the pre-existence of pain in the spot which has spontaneously broken. In such cases the disease of the bone, instead of producing a mass of cancer, may be found in the form of a cyst-like hollowing out of the shaft, containing a pulpy, gelatinous red matter, with the histological structure of sarcoma, or sometimes more closely resembling a localised patch of true osteo-malacia.

But the bones may break spontaneously through mere *fatty degeneration*, though we cannot say that such an occurrence has come under our own notice. After thus breaking, it is remarkable that they may show a power of repair sufficient to heal the fracture, and it is still more surprising that, even in cases of cancerous fracture, repair has nearly succeeded in some instances. Lastly, in *violent efforts* perfectly healthy bones may be spontaneously broken, *i. e.* broken by the action of the muscles that naturally move them. This is especially the case with the patella and olecranon.

Rickets.—The bones are, as you know, first represented by cartilage, which changes into bone by an interesting and curiously complicated process. This process, described shortly, consists of a multiplication of the cartilage cells, a calcification of the cartilage when rendered richly cellular through this multiplication, a formation of vacuolæ in this calcified material, and a production of true bone tissue within these vacuolæ. Although this process is so complex, yet, when all goes well, the area in which it is in course of proceeding is almost microscopically narrow. The completion follows the beginning of the change so quickly in the line of advancement that only a very thin layer of cartilage is found undergoing ossification at any period; and this layer forms properly a very even and local separation between the bone and the cartilage. Further, within this narrow advancing line the successive stages of the change are equally orderly, from the cartilage, through the before-mentioned modifications, layer by layer, to the bone beneath. When a part of the original cartilaginous shaft is converted to bone throughout all its thickness, consuming the cartilage until the bone reaches the periosteum, then the further growth of the bone in thickness is effected by the ossification of the deep layer of the periosteum. This is a much simpler process, which consists, shortly, in a simple calcification of the matrix, and transformation of the cells of the periosteum into bone-cells.

You will easily see that since the shafts of long bones are comparatively early converted to bone, while by their aftergrowth they become so large that the young bony shaft could be entirely lodged within the marrow canal of the adult shaft, it follows that, practically, the whole of the adult shaft is formed from periosteum. We need not remind you also that the bones of the vault of the skull are formed directly out of membrane without cartilage.

Now, the intermediate matter which forms the stages of transformation of cartilage into bone, is of softer consistence than either cartilage or bone; and the same may be said of the substance which is intermediate between periosteum and bone; it is softer than either (hence the ease with which the periosteum may be stripped from the bone; such stripping is a tearing through of the deeper, softer, ossifying periosteum). From this it is easy to see why the amount of intermediate matter naturally present at any time should be small; for if a great quantity of it were present, the bones would be liable to bend or to break along the growing line, especially as the formation of the new bone on the outside is always accompanied by a removal of the older, deeper bone from the inside by the development of cancelli and of the medullary cavity. For as the shaft grows outward, the medullary canal is always increasing in the inside.

The disease known as *rickets* consists of a disturbance of the process of ossification, such as to delay its ultimate stages, while it hastens the earlier; thus producing a mass of imperfectly ossified matter upon the growing end of the young bony shaft; while it also softens the shaft itself, by making a quantity of incomplete and weak bone under the periosteum. Meantime the medullary canal, widening, removes much or all of the better bone formed before the rickets set in. Thus the bone becomes thick, soft and weak, its ends being swollen through the presence there of the actively growing cellular mass of disordered ossifying cartilage.

The microscope shows the disease to consist of an intricate confusion of the order of the complex ossifying process, without any very wide departure from its essential nature. The multiplication of the cartilage cells progresses to an excessive extent. The calcification which should immediately succeed more or less fails. Yet the formation of medullary vacuoles occurs in the unossified mass, and, meantime, some of the descendant cells of the cartilage undergo interesting changes approximating their character to that of bone-cells; the interior acquiring a stellate figure resembling that of a lacuna. Thus we find, mingled together in chaotic confusion, crowds of multiplied cartilage cells, medullary spaces, and calcified patches, with various forms and degrees of transformation from the cartilage to the bone-cell.

These, heaped up, make the soft and swollen mass at the end of the shaft between the bone and cartilage. This soft mass is more vascular than normal, being permeated by numerous loops of vessel which run on often some distance into the cartilage itself. So, too, the periosteal layer of bone is thick, soft, and vascular, and the whole bone is everywhere congested, the contents of its medullary cavities being unnaturally red, while the fat disappears from them. In severe cases the connective tissue around the bone and among the adjoining muscles may take part in the process and undergo more or less swelling, so that the disease comes to wear some of the characters of an inflammation.

Much constitutional disturbance attends these more severe degrees of rickets; so that the child may die with broncho-pneumonia, wasting diarrhœa, &c., if the case be acute or grave. But when the process s of medium severity the local effects are those that chiefly command attention. The softened bones yield under the action of the muscles or the weight of the body, so that they become curved in bad cases to a surprising extent. The process of growth is also inefficient, so that they become shortened, and thus the stature is stunted. The more pronounced curvatures are of great importance, especially those of the pelvis and ribs. These we shall describe when speaking of alterations of form in the skeleton. The curves will be observed in milder cases to be exaggerations of the natural slighter curvatures present in the bones, so that little or no deformity then results.

The process of rickets belongs only to the stages of ossification, passing off long before their completion. As the rickety condition disappears the bones undergo changes of an opposite kind. The thickened soft spongy substance becomes filled up with hard bony matter until the shaft is made very dense and solid, but permanently deformed by the curvatures that were suffered during the active stages of the disease.

Mollities Ossium, or Osteomalacia.—This differs from the disease last mentioned in being one which affects the adult, and in most instances has come on in women during pregnancy; this, however, is by no means always the case, as the disease occurs in men, and in persons of both sexes at advanced periods of life. There is, as in rickets, a preponderance of the animal over the earthy constituents, the latter gradually being lost, and having been observed in many cases to pass off by the urine; but, unlike rickets, the disease does not generally stop but progresses in most cases to a fatal termination, during which time the patient is bedridden, and the different parts of the body have a tendency to fall together, the body becoming bent like a bow, and the limbs drawn together, and capable of being bent in all directions. The bones are so soft that they may be cut with a knife, and so light that they float on water, as in the case of these vertebræ. The earthy matter is all but gone, and nothing but the gelatinous part remains.

The disease shows itself first in those bones which have but little compact substance, such as the pelvis, vertebræ, ribs, &c., and later in the hard shafts of the femur, humerus, &c. In all cases it tends to spread and involve the whole skeleton.

To the naked eye the disease appears as a widening of the medullary spaces at the expense of the bony tissue, the contents of the spaces becoming red and gelatinous, while the trabeculæ and plates of the spongy bone grow thin and soften, until many disappear, losing their lime salts first, so that they can be cut with a knife or bent like horn. The microscope does not reveal much in explanation of the process; we find the bone-corpuscles altered in form, losing their fineness of outline and regularity of appearance, so that they become widened and irregular in shape, and appear to have fewer canalicular processes; meantime the medulla is congested intensely, and is found to contain an excessive proportion of lymphoid corpuscles such as are naturally present in smaller number, while its fat generally disappears. But at what appears to be a later stage some cases show less of congestion, the cancelli containing a white gelatinous or fatty degenerate matter.

The specific gravity of this vertebra is scarcely ·7, while healthy bone is nearly two or three times as heavy. The bones may bend and subsequently unite, as seen in these ribs. The scapula may become thin as paper and doubled upon itself, so also may the os ilii and sacrum.

Alterations in Form of Head, Spine, Thorax, and Pelvis.—Instead of speaking of the changes which take place in the various bones under the different forms of maladies which cause them, it will be more convenient to review the deformities of the particular parts of the skeleton at the same time, so as to compare them together.

Head.—The most remarkable alterations of the skull are affected by *hydrocephalus*. In this disease the fluid in the ventricles of the brain expands the head, and the bones of the calvaria are widely spread and separated. Such skulls offer several characteristic peculiarities. There is, first, a great disproportion between the enormous size of the cranium and the comparatively very diminutive facial bones below it. The vault is very thin, and for a long time is in part membranous, and the indentation of the sutures is deep. The supra-orbital ridge is done away with from the stretching upwards and forwards of the frontal bone. If the child live and the fluid is not absorbed, fresh points of ossification arise, and thus supplementary bones grow in the interstices of the natural ones. These are of various shapes and sizes, and are named ossa triquetra, or Wormian bones. You will see such bones of very great size in this remarkable hydrocephalic head of the man Cardinal, which measures thirty-three inches in circumference and held a gallon of fluid. You will observe that the form of the orbit is very remarkable in hydrocephalic crania; the roof of it, instead of being horizontal, is inclined more to be vertical, and this gives the eyes the peculiar roll so characteristic of hydrocephalus; at least, the peculiarity of the orbits is found in all the infants' skulls before us, though it lessens in those who live to be older.

In *rickets* also the cranium is disproportionately large and often overhanging in front, but a rickety cranium does not afford such evident characteristics as those which belong to hydrocephalus. The sutures and fontanelles are late in closing, and during the progress of the rickets, which may be at any time from birth to the tenth year, the bones are light and thin and porous, being hyperæmic when fresh. They may also show gaps in their continuity along the course of the sutures, especially the lambdoidal, these gaps being filled in with membrane; but afterwards, when the disease is healed, the opposite condition establishes itself, and the bones are found dense and thickened along their edges, while their middle parts may be either thin and hard or natural, or perhaps very thick; but it is still a question whether the great extent of porous thickening of the cranium we have before described is due to rickets or not. It is certainly sometimes associated with it, while it is equally certain that great rickety deformity of limb may be found with a normal cranium.

Our museum also presents some other curious specimens of malformed and unsymmetrical skulls. In this you will see the sides flattened and raised up in ridges towards the top; in this one you will see the sides are not alike; this is a skull of the flat-headed Indian, the front part of whose head was thus flattened in infancy by artificial pressure.

Spine.—Angular curvature, i. e. the bending of the spine to an angle projecting backwards, is called *kyphosis*. It results from disease and destruction of the bodies of the vertebræ and their consequent falling together; this occurs mostly in the dorsal region; indeed, caries of the lumbar and cervical spine do not often produce this curvature.

Where the bodies of several dorsal vertebræ are destroyed the curvature of the back is excessive, as in this case, where the nine dorsal and three upper lumbar are excavated away, and their remains form a small angular mass, to which the transverse processes and ribs are attached. In some cases the remains of the vertebræ are held together by ligamentous tissue. The cord is bent and follows the inequalities of the spine; and it is remarkable to what extent the bones may be affected without the cord participating.

Lateral curvature.—When the spine is bent in a curve to one side the term *skoliosis* is used. A slight curvature of this kind is very common, especially in women; but there seems no limit to its extent until, indeed, the spine shall be bent down parallel with itself, as is seen in this very remarkable specimen, where the ribs actually pass down in front of it. Skoliosis does not result from caries of the spine like angular curvature. Its cause is, indeed, comparatively obscure. Lesser degrees of it are found in rickets, but the more severe examples are found in persons who do not show any of the characteristic results of rickets, such as peculiar bends of the long bones, enlarged epiphyses, &c. It will be seen, on examining this and other specimens of lateral curvature, that the vertebræ themselves are not much altered in shape, but the bend is due almost entirely to the wedge-like form of the cartilage between them, this being wide on the convex side and narrow on the concave; on the latter the bones actually touch, and in some cases are superficially ankylosed, and so likewise in the concavity of the bend the heads of the ribs are so crowded together that ankylosis may take place both to the vertebræ and to one another. By regarding attentively a spine thus laterally curved you will see that the vertebræ have suffered a rotation on their axis, or have so turned on themselves that the bodies of the vertebræ form the prominence of the convexity, as will naturally be the case if a spine be forcibly bent downwards, while the spinous processes are turned to the concavity, one set of transverse processes projecting backward. One side of the chest is necessarily carried backwards in this rotation of the spine, and thus the hump on the back is formed by the angles of the ribs as well as the transverse processes of vertebræ.

In some cases, as in rickets, the spine is so curved that the lumbar vertebræ, with the promontory of the sacrum, project much forwards, and the back necessarily falls in. This curvature is called *lordosis*.

In *mollities ossium* the tendency is for the head and pelvis to fall together, and the spine thus makes a long curve backwards in the opposite direction to that in lordosis.

Thorax.—In lateral curvature the thorax is necessarily much distorted, one side being narrowed and the other widened. By looking at this preparation you will see what generally occurs. The spine is here bent so that the convexity is on the right and the concavity on the left; as we have already seen, the ribs on the convex side are carried backwards by the rotation of the spine, which always makes the bodies form the front of the curve. Thus the ribs and transverse processes together form the prominence of the hump-back. The ribs so carried back have necessarily to run a longer forward course to the sternum, which retains its natural position. To do this they are stretched out straightly, so as to make the antero-posterior diameter of this side of the chest great and its lateral diameter small. On the other hand, the ribs on the concave side, being necessarily carried forwards by the twist of the spine, have a correspondingly shorter distance to run to the sternum, so that they dispose their length in a stronger lateral curve, and thus widen-out that side of the chest, while its vertical depth is, of course, smaller than on the other side. Thus it comes about that in skoliosis the lateral measurement of the concave side of the chest is great and the antero-posterior and vertical measurement small, while, on the convex side, the lateral measurement is small and the antero-posterior and vertical great. The ribs on the concave side may meet each other and become ankylosed, especially near the spine. The sternum keeps its natural position in the median line. In slighter lateral curvature the chest may be bent a little to one side with the spine, without much alteration of form. On the other hand, when there is a double curvature the alterations may be so complex that it is scarcely possible to describe them. The usual condition in lateral curvature is, however, such as we have stated above. In *angular curvature* or *kyphosis* the chest is symmetrical, but bends down towards the pelvis, so that, as you see in this specimen, the lower ribs reach below the ilium. The vertical measurement is decreased, while the antero-posterior is considerably increased and the sternum is carried forward, so that, as you see, it is in a vertical plane considerably anterior to that of the face.

In *rickets* the thorax presents a characteristic kind of alteration, corresponding to what is popularly called "pigeon breast," a name given to it from the prominence of the sternum. In this disorder, as we have already said, the bones are soft, especially the growing ends of the bones. Hence the ribs do not firmly and equally resist the atmospheric pressure in the act of respiration, as they normally should, but yield, so that the softer parts recede. Thus it comes to pass that the line along the thorax, which corresponds to the softened ends of the ribs, sinks in, forming a groove on each side, giving relative prominence to the sternum. This groove on the left side is shallower on account of the presence of the heart beneath. On the right side it is deeper until you reach the lower part, where the liver lies beneath supporting the ribs. Of course you will see that this effect of atmospheric pressure will be more evident if the entry of air into the chest is not free, since then the breathing efforts will be more forcible and the suction greater. Indeed, in some cases, a similar "pigeon-breasted" state of chest may come about without any evidence of rickets, being produced through any of those causes, such as enlarged tonsils, small trachea, capillary bronchitis, &c., which permanently obstruct the entry of air.

On the other hand, when the original expansion of the lung substance is imperfect, so that a part of it remains airless in the condition called *atelectasis*, or when through *whooping-cough*, &c., parts of the lower lobes are shrunken permanently, so as to diminish the size of the lower parts of the lung, the lower part of the chest is shrunken in comparison with the upper part. This contrast between a large upper and a small lower part of the chest is increased by the occurrence of compensating expansion of the upper part of the lungs such as we shall describe.

In *mollities ossium* the parts of the chest have a tendency to fall together, as you see in this specimen; the ribs are softened, some fractured and bent into various shapes, and the chest falls in at the sides. The spine is curved, forming a convexity backwards; and the sternum is often, as you see here, bent in its middle.

In *emphysema* the thorax is permanently in a condition of extreme inspiration; indeed, more expanded than in ordinary inspiration in a healthy chest. The lungs having lost their elasticity, the act of inspiration is only effected by muscular effort and the recoil of the ribs—processes which naturally constitute only the earlier, if the greater part, of the expiratory act. This act is never finished as it should be by the elastic recoil of the lung, and fresh breaths are taken into the lungs already partly full, until the muscular effort required to renew the air is so great as to largely increase the curvature of the dorsal spine. For the inspiratory muscles draw down the upper part of the spine in raising the ribs, and the expiratory muscles draw up the lower part of the spine in depressing the ribs, and the spine above is drawn downwards by the first and upwards by the second, so that in this way the dorsal part of it becomes bowed backwards in the way so characteristic of old asthmatic or emphysematous subjects, thus increasing the antero-posterior diameter of the chest. Such subjects are also high shouldered, for, of course, the shoulders go up with the chest. Other changes may occur in the chest from disease. Thus, from effects of pleurisy, one side may be contracted, and even the spine be bent; and, from the same cause, a local contraction or depression may arise at the lower part of the chest, or at the upper part beneath the clavicle.

Pelvis.—This may be variously misshapen. First we shall consider the effects of rickets and osteo-malakia. The respective effects of these on the pelvis differ almost characteristically. In *rickets* the sacrum and lower lumbar vertebræ are bent forward (lordosis), so that the former approaches and almost meets the symphysis pubis, which is pushed upwards to meet it. The cavity of the pelvis is thus narrowed from before backwards, especially in the middle, while it is elongated from side to side, thus, on the whole, resembling somewhat the figure of an hour-glass. It is not difficult to understand how this shape is produced if you recollect that the growing edges of the bones are the soft parts in rickets, and that the growing edges are in the acetabulum, the sacro-iliac synchondroses and the symphysis pubis. Those being the soft parts, and the thrust being made from the spine to the thigh bones, so as to squeeze the pelvis between, you will see that the femoro-vertebral diameter will become narrow, the softish acetabula giving way towards the spine, thus flattening the pelvis from before backwards and producing the hour-glass shape when the symphysis goes with them, but in some cases of rickets the hour-glass shape is not so evident, for the symphysis pubis projects forwards, making the inlet of the cavity triangular. In *osteo-malakia*, or mollities ossium, the softness of the bones is equal in all parts, and hence not only is the sacrum bent downwards, but also in bad cases the bone about the acetabulum on each side is thrust upwards towards the sacrum, leaving the symphysis pubis projecting like a beak. The bones, even the ala ilii, bend freely whilst yielding, thus producing an appearance which is very surprising in comparison with the usually hard and incompressible state of the pelvis. On the other hand, in the rickety pelvis, though the front of the pelvis with the pubis is pushed backwards towards the spine, yet there is no bending of the other pelvic bones.

In *angular curvature* of the spine the pelvis is generally deep, like the thorax, as you see in this specimen.

In *lateral curvature* the pelvis generally becomes slightly distorted, owing to its sinking down on the side of the curvature. In this

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specimen you will see that on the side of the convexity the pelvis is raised, so that the tuberosity of the ischium on this side is higher than that on the other, and the pelvis is necessarily distorted.

The *oblique pelvis* of *Naegelé* is due to ankylosis of the sacro-iliac synchondrosis occurring in infancy, or even in intra-uterine life, so that during development a distortion naturally ensues. We may look upon this as a specimen of the affection. You see the right sacro-iliac joint is ankylosed and the pelvis is unsymmetrical. The disease of the joint has been accompanied by much loss of substance to the ilium and sacrum on the right side, and thus the latter bone is thrown in this direction, and the whole right side of the pelvis is smaller than the left. Thus, the front of the sacrum does not face the symphysis pubis, and if the diagonal measurement be taken from this diseased joint to the left acetabulum on the opposite side, it will be seen to be greater than that taken in the other diagonal.

Morbid Growths.—The tumours that form from bones present a general resemblance to the tumours that form in the soft parts. But in accordance with a rule which we shall frequently have to mention, by which tumours have more or less of the structural character of the tissue they form in, the tumours of bone are more or less like bone, the resemblance being generally greater in proportion to the slowness of their growth. Such resemblance was thought to be due to some obscure influence the surrounding tissue had on the development of the newly formed material. But there is great reason to think that the new material of the tumour is derived from the bone by a direct multiplication of some of its structural elements; so that the likeness of the tumour to the matrix, or part it grows in, is an hereditary likeness due to descent. But, as we have before had occasion frequently to remark, a whole bone consists of several tissues, each distinct in character from the others. Thus there are at least these: cartilage, periosteum, medulla, tela ossea and vessels.

A new formation from bone may take its rise in and its characters from either of these components; so as, in short, to be cartilaginous, or fibrous, or myeloid, or osteoid, or vascular. When the growth is slow the character of either of these tissues is tolerably well observed in the tumour; and then generally the tumour is "innocent," or devoid of those infectious properties that constitute malignancy. But, if the growth is rapid, the new formation does not acquire the special characters of the tissue it arises in. Its elements, hastily produced, show much of their original round unspecialised shape, or have within them, in the form of large nuclei and nucleoli, evidences of their vivid reproductive activity.

Besides their liability to primary tumours, which is relatively considerable, the bones are also not unfrequently the seat of secondary cancer, which, indeed, may be widely distributed in the skeleton, as we shall see. The tumours which most commonly arise in bone are—

Osteoma.—This name is given to such tumours as are *practically* composed of bone. When they grow from the surface of a bone they are called *Exostoses;* when they form in the interior of a bone they are called *Enostoses.* The relations of exostoses to inflammatory nodes, &c., on bones have already been considered. We must remember that the *tela ossea* or proper hard substance of bone is mingled always with certain soft tissues, from which it arises and by which it is supported. It will be readily anticipated that pathological new bone, formed as it is under abnormal conditions, and without the harmonising influence of natural development, will often show a disproportion of the several constructive elements found in healthy bone. Thus we sometimes find in bony tumours an excessive development of the tela ossea, so that the bone becomes as hard as ivory, and almost entirely devoid of medulla, and even of vessels; such tumours are called *Ivory exostoses.* Their favorite seat is about the face and anterior part of the skull near the face, especially near the frontal and other sinuses, where they form more or less rounded nodulated tumours of extreme hardness. The ivory change appears sometimes to go on until it destroys the life of the tumour by stopping circulation in it; and so in this remarkable case, described by Mr. Hilton in the 'Guy's Hospital Reports,' the tumour may spontaneously separate. This form of tumour has often been observed to arise within the diploë of the skull in the frontal region, growing and separating the tables from each other, until it bursts through one or both of them. Such an occurrence you see in this case of Mr. Birkett's, also described in the 'Guy's Hospital Reports.' It is for such a production of bone that the term enostosis is used.

Other exostoses show natural cancellous tissue, and occasionally one arising from a long bone contains a marrow canal in communication with that of the shaft of the bone.

But bony tumours do not only vary in the relative density or rarity of the bone that composes them, for they also offer a considerable variety in the amount of the associated soft tissues—cartilage, periosteum, medulla, &c.—which accompany them in a more or less modified state. Thus many bony outgrowths have a covering of cartilage, which forms their growing part, and from which the bone develops in the growth of the tumour. In such cases it is not easy to decide whether to call the tumour an ossifying enchondroma or a cartilaginous exostosis; but this difficulty is only verbal. It is practically settled by calling the tumour after the constituent that enters most largely into its composition. On the femur such tumours are not infrequent. Facts have been adduced to show that such cartilaginous exostoses affect most frequently the situation of the epiphysial cartilage as though they took their rise from that. But when we remember that, in the reproduction of bone after fracture, or in the production of bone by transplanted periosteum, a cartilaginous stage is often passed through, so that periosteum can thus be made to change itself into cartilage, there appears no occasion to go far to search for a reason why exostoses should be tipped with cartilage. Such an exostosis, further, may have a bursa over it, and the sac of this bursa may communicate with the cavity of a joint; when exostoses grow near a great joint this is a possibility that must be carefully weighed by the surgeon before operating. In yet other cases the new growth of bone occurs within a great mass of a tissue more or less resembling the periosteum, and this tissue sometimes is more like fibrous tissue, and at other times more resembles cartilage. In such cases it is often quite a question of words whether the tumour should be called an osteoma or an ossifying fibroma, when the ossifying tissue is fibre, or an osteoid chondroma, when it has a structure resembling fibro-cartilage. But in the latter case the tumour is of a more doubtful and suspicious character, so that it is more important to make the distinction; many very large, dense, bony tumours are of the nature of osteoid chondroma.

In short, there are not many osteomata or tumours of pure bone above the dimensions of exostoses or mere bony outgrowths. The greater tumours, which contain a large proportion of bone, show along with it an important amount of some one or other of the allied tissues, such as periosteum, cartilage, medulla, &c., whose subsequent and final changes end in the increase of the bony mass by continuous transformation of their tissue.

Enchondroma.—The bones are the favourite seat of enchondroma, and those enchondromata which occur in bone are generally more purely cartilaginous in their structure and more innocent in their tendency than the rarer enchondromata which arise in the soft parts; for instance, in the testis. Virchow has advanced the belief that *tumours which in structure resemble the part they grow in* are generally of an innocent nature; and remembering that cartilage is nearly always united with every bone, and forms a stage in the development of most bones, its presence in bone is not a great departure from the nature of the parent tissue; so that thus, on Virchow's view, an enchondroma in a bone comes near to possessing that local homology which he regards as the criterion of innocence in tumours. On the older and simpler view of homology which considered as homologous and innocent *any tumour whose substance was like any texture of the body*, wherever it might grow, the cartilage tumours of bone are the very purest type of homology and innocence. Their histological structure departs very little indeed from that of ordinary permanent cartilage.

But the usual seat of an enchondroma in a bone is where cartilage is not naturally present, as within the shafts of the long bones and phalanges, or on the surface of the flat bones. Virchow suggests that an included portion of unossified cartilage remaining from former rickets may explain this, but no facts are adduced to show the actual truth of this view. The bones of the limbs are liable to cartilaginous tumours in proportion to their distance from the trunk. The tumours are often multiple; thus they may affect many bones of the hands, and produce enormous deformity. They are observed to arise either within the bone or on its surface, and have been divided accordingly into *peripheral* and *central* enchondromata. Those of the long and short bones generally arise centrally, and those of the flat bones, as the pelvis and scapula, usually arise peripherally. In the former case they expand the shaft of the bone to a mere shell, which, spread over them, holds their surface into some smoothness and evenness, while in the latter case the tumours project freely and have a more uneven surface, from the separate freedom of the lobes of the tumour. Enchondromata are of a lobulated structure throughout, so that, on section, one sees the clearer bluish cartilage divided into areolæ by plates and beams of a more fibrous whiter substance, into which vessels extend. The cause of this lobulated structure appears to be the increase of the tumour by the starting up of fresh centres of growth in the tissue on the borders of the main mass, which then grow and coalesce with it. Where one of these enchondromata is irremovable from its position, so that it grows very large, as in the scapula or pelvis, it is apt to soften in parts to an almost gelatinous consistency, or even in places to melt down as it were and form cysts. It is then called *cysto-enchondroma*. Some enchondromata have cysts that are not explicable in this way. The increase of the tumour may stretch the teguments until they ulcerate, and some portion of the tumour may break down and be discharged; producing, from the size of the mass, and the great excavation in it, a frightful degree of misery.

In one case of Mr. Birkett's, which we inspected, the tumour found in the scapula weighed many pounds. Besides the softenings they undergo, enchondromata vary in in their consistence. It is generally admitted that those which are softer are of less benignant disposition. Their microscopic structure then approaches that of myxomata, and they are called *myxo-enchondroma* (see Pl. III). Some of the softer enchondromata have been known to recur on removal, as in the remarkable case from which this preparation was taken. The tumour occupied the upper jaw, and after growing nine years was extirpated by Mr. Morgan; it sprouted again, so that at the patient's death, seven years after the operation, it had reached the immense size seen in this cast. Other examples of enchondroma have proved infectious to remote parts. Thus an enchondroma of the scapula has been associated with similar disease in the lungs.

We may here mention the occurrence of small outgrowths of cartilage called *ecchondroses* from the permanent cartilages, such as those of the ribs; ears; symphyses, &c. In these situations they have not reached any important size; but in the larynx they have been known to project as polypoid growths from the cartilages. Such ecchondroses of the articular cartilage may take part in diseases of the joints, and will be mentioned in that connection.

We have several times met with a soft mucoid mass under the pons Varolii, between it and the basilar bone. It is composed of delicate round cells. In the cases we have seen the growth did not appear to have exercised any pressure; it was evidently the same thing as was by H. Muller referred to a persistence of the top of the fœtal notochord, and which Virchow has proposed to call *chordoma.*

Osteoid chondroma (osteoid cancer).—This name, which we have already several times used in connection with fibroma and osteoma, is one that very well expresses the structure of the soft growing part of an important kind of rapidly ossifying tumour, which often grows to a large size, and which also has a serious interest from the tendency which it undoubtedly has to malignancy. So many recurrent ossific tumours have been recently found to be of this kind, that it is doubtful whether most of the so-called "osteoid cancers" of authors were not really osteoid chondromata. They form dense, hard, heavy tumours, a large part of which is nearly always composed of bone springing from the periosteum, and also extending inwards to fill up the medullary canal, thus obliterating the outline of the original bony shaft. They grow very frequently on the articular end of a long bone, especially about the knee-joint. The tumours they form are tolerably uniform in outline, but have lobes of low elevation separated by grooves in which vessels, nerves, &c., are lodged. The structure is a dense whitish fibrous tissue, grating on section, and having a close tendon-like texture. The microscope shows a structure which can best be compared to those plates of osteoid cartilage which are so common on the spinal pia mater after middle life, *i. e.* it closely resembles bone which has been decalcified by acids, and its transformation into bone is simply effected by the addition of lime salts to the matrix between the cells, which themselves become bone-corpuscles (see Pl. I). We recently had under our own care a man with a very large tumour of this kind at the upper end of the humerus. In the course of his disease painful paraplegia came on, and after death there was found an exquisite example of the same osteoid chondroma in the middle dorsal vertebræ, and a great number of similar tumours were present in the lungs. It is this tendency to metastasis which earned for it the old name osteoid cancer. The name osteoid chondroma is preferable, as it refers to the recent and active part of the tumour in which its dangerous activity lies, for when once the mass has ossified it remains stationary, the morbid bone being, so far, as innocent as any natural part of the skeleton. The mischief runs on through the production of the new-formed cartilage in the new exterior advancing part of the growth. Attention must always be mainly directed to the active growing parts of tumours.

Fibroma.—Fibrous tumours are not often found arising from bones; most examples recorded or preserved in museums have affected the jaws. Some have grown within the bone and expanded it. Others arise from the periosteal surface, especially that of the alveolar border of the jaw, so that the tumour in its growth raises the gums, whose tissue is spread over it, thus making one sort of "Epulis," for by this name any tumours of the gums are called. Such fibrous tumours are generally permeated by a more or less dense framework of bone, which extends from the affected part of the jaw, and spreads in dendritic form through the growth, continually increasing by ossification of the fibrous tissue (osteo-fibroma). Pure fibromata are very rarely found anywhere else on the skeleton. We have one specimen on the phalanx of a finger, and one has been occasionally met with in other parts. But although tumours of pure fibrous tissue are thus rare, yet fibrous tissue is found not infrequently forming considerable parts of mixed tumours, such as *fibro-enchondroma* or *fibro-sarcoma*, wherein strong and broad bands of fibrous tissue pass through the tumour, dividing into lobes, and composing a greater or smaller part of it. That fibrous substance which is present in many large growths from bone, which are called osteoma from the prevalence of bone in them, is often mistaken for mere fibrous tissue, when closer examination will show it to have the structure we have just described as osteoid chondroma.

Sarcoma.—When considering sarcoma in comparison with fibroma, osteoma, enchondroma, &c., we observe that sarcoma shows this important difference from those classes of tumours, that there is no standard normal tissue to which we can refer its structure for comparison. There is no normal complete tissue like in structure to sarcoma.

The essential feature of the histology of sarcoma is the presence of cells in an intercellular tissue, forming a homogeneous web. The cells are found to vary very much. Thus, some are spindle-shaped, large or small, others are round cells, large or small; and, besides, there are found in some sarcomas cells with many nuclei; they are very large, and generally indented at their edges by the pressure of their neighbours. These are known as giant cells from their size, or myeloid cells from the presence of similar cells in fœtal marrow. For the varieties of cells in sarcomatous tumours see Plate II.

The sarcomas of bone are conveniently divided into those which arise externally and those which arise internally, or *periosteal* and *endosteal*.

This division does not separate all of them clearly into two groups, since it is often difficult to say whether the tumour arises from periosteum or medulla, yet it constitutes a convenient division, for, speaking generally, the periosteal and endosteal sarcomas show characteristic differences each from the other.

The *periosteal sarcomas* form tumours whose consistence varies from a fibrous toughness to a fleshy or even tremulous softness, and they are, as usual, soft in proportion to the rapidity of their growth; they have an elastic consistence, and do not yield a milky juice when scraped. The surface of the tumour is generally in bold convexities, either incapsulated in fibrous tissue, which it is unable to infect, or if it be more diffusive and infectious, extending into the muscles, &c., which it is meantime pushing before it in its growth. Such a tumour is very liable to ossification of its structure. The new substance takes its rise close to the bone under the periosteum in the deep layer of that membrane which Müller called its "cambium layer." In its growth it elevates the periosteum off the bone, and in some instances the periosteum of the marginal parts of the tumour thus displaced undergoes ossification in some points of its new position, forming a layer or layers of bone at an angle with the shaft. When it has grown large such a tumour will nearly always offer a considerable variety of appearances in different parts. Blood will often be effused in some places, and when found in various stages of decay will offer different shades, from red, through brown, to pale yellow cheesy colour; or cysts will be present in the tumour, arising as apoplectic cysts from hæmorrhage, or else from softening down of the tissue to form cavities, or sometimes by active development of cystic spaces lined by epithelium. The tissue of the growth itself may often differ in different parts, some parts having a somewhat mucous or glutinous consistence from the presence of some mucin in the intercellular substance (myxosarcoma), while others are tough and fibrous, showing a quantity of pure fibrous tissue in their composition (fibro-sarcoma), in others yet more or less extensive patches of cartilage are found (chondro-sarcoma), while in others, and this frequently, there is a large development of bone ramifying through the mass (osteo-sarcoma). The bundles of fibres and cells in the mass wind about irregularly, and the section dividing them at different angles, will cause a faintly mottled appearance, while, lastly, the fatty decay of portions of the growth often produces more obvious opaque mottlings. All these appearances may be found in a single tumour; more commonly they are not all present, but there is a prevalence of one of the varieties of composition described, and from this prevalence of or purity of one variety, the tumour would be called fibro-sarcoma, myxo-sarcoma, chondro-sarcoma, osteo-sarcoma, cysto-sarcoma, hæmorrhagic sarcoma.

The interior of the bone at the affected part may undergo simple induration only, the excess of supply in the close neighbourhood of the tumour inducing excessive growth; or else, and in many cases indeed, the tissue of the tumour is found not only outside, but also in the interior of the bone.

Sarcomatous tumours are found more frequently near the articular ends of the long bones than elsewhere in the skeleton. They do not implicate the joints, at least, only in very rare instances, which we never met with ourselves. A remarkable case is, however, given by Dr. Steudener, of Halle, where a round-cell sarcoma, reaching the knee-joint, grew freely in the synovial membrane, and in that only.

The bony development in *osteo-sarcoma* may reach a curious degree of complexity, forming, when prepared as a skeleton, a number of beautiful spiculæ, proceeding from all around the bone, and branching as they extend, to terminate in an even coral-like surface in the outer part of the growth. The bony skeleton of the tumour is generally stout and firm, in proportion to the firmness of the tumour, and that is proportioned to the slowness of its growth, as we have, indeed, already said.

At first the periosteal sarcomas are kept in by the periosteum, but soon the rapidly growing examples find their way through this membrane and spread in the tissue outside, into and among the muscles generally, in bold, advancing, round masses of the flesh of the tumour, which may be seen partly destroying the muscle by coarse pressure, and partly transforming it by spreading minutely along the interstitial tissue between the fibres, and so crushing these out of existence.

The histological structure of the periosteal sarcomata shows in different instances, or perhaps in different places in the same instance, all varieties of the proper structure of sarcoma, that is, a homogeneous tissue of round or spindle-formed cells, large or small, with more or less of an intercellular tissue, connecting them into a web. Sometimes there is a small proportion of giant cells, but not many, and doubt has been raised whether the examples with giant cells are not in reality developed from the medulla rather than the periosteum. Besides thus varying in the kind of sarcomatous tissue present, there are further and wider varieties of microscopic structure corresponding with those variations of the tumour into myxoma, enchondroma, osteoma, &c., which we have before mentioned; but, beyond saying that such varieties show all the microscopic characters of these several kinds of tumour, and all stages of intermediate growth between them, we need not pursue this subject, but will refer to the figure in Plate II.

Although the periosteal sarcomas are broadly distinguished from the sarcomas that arise in the medulla, yet, as we have already hinted, many periosteal growths show so much coexistent endosteal formation that it is difficult to decide whether the outside or inside of the bone is most affected.

Endosteal sarcomas, myeloid tumours.—Those tumours which arise in the medullary tissue and distend the bone have always created more surprise than the kind we have just described. Indeed, when we find the shaft of a bone, rigid as it naturally is, widened so as to form a great bony sac, there is reason for wonder, and some observation and reflection are required to explain the change; such expansion of the bone arises in this way. While the pressure of the growth within removes the old bone by absorption, it presses out the yielding periosteum, which, while being very gradually thrust into a new position, continues to make new bone as it goes, thus keeping a shell of new bone more or less continuous when the process is sufficiently slow. But, on the other hand, if the process of expansion is very rapid, there is no time for such ossification, and the whole bone is softened away at that part by the growth, so as to seem to cease abruptly as it enters the tumour. When the substance of the tumour is removed from the interior of such a bony sac, as when the bone is put up in the form of a skeleton preparation, the effect is very remarkable, and all museums contain some such specimens under the old name *spina ventosa*. The kind of endosteal sarcoma which thus distends the bone is in the form of a growth, often having a peculiar deep crimson-red colour, and being soft and dry, not at all fibrous, and yet not yielding any juice on scraping as cancers do. The red hue of the substance is very peculiar; it formerly gave the name *spleen-like* to the tumour; it is somewhat like plum juice, and is different from the colour of any natural tissue of the body. There is often some blood extravasated in the tumour, which may increase the redness; but when this is removed the substance itself is found to have this red hue. In some cases the tumour is soft throughout, in others processes of fibre proceed from the outer surface, and split up into a reticulum more or less dense, constituting thus a *fibro-sarcoma;* in other cases cysts are formed in it. The most usual seat of such tumours is the lower or upper jaw, and the next most frequent seat is the end of a long bone. Beginning in the centre of the bone, the growth continues until the shell of bone is reached and lifted, as already described, the process in its spread encounters at last the articular cartilage, but this generally is not involved, and in this specimen, which was taken from a young subject, the epiphysis is not involved. The microscopic structure long ago arrested attention from the peculiarity it has in the great proportional quantity of very large "giant cells," which interestingly resemble the large polynucleated cells of fœtal marrow, suggesting a return of the fœtal type in the growth of the tumour. These bodies have been called *myeloid cells,* and the tumours themselves were hence called myeloid, when it was not sufficiently recognised that these large, many nucleated cells are very often present in morbid growths, and are not limited to the tumours which occur in the medulla of bones, but are also found in myxoma, and in lymphoma, and even in tubercular formations, they are now known only as *giant cells* (see Plate II, *b*). However, in the "myeloid" tumours we are describing these cells are much more plentiful and better formed than in the other tumours we have mentioned. They are surrounded by intermediate cells, generally of the usual spindle-form type. Their large proportion of giant cells, together with the peculiar colour of the growths, mark them as a distinct variety. Although these giant-cell tumours generally arise within the bone, yet in one of their favorite seats under the gums on the jaws they show a more superficial connection with the bone, being often united with it by dense bone; in these examples it is difficult to say whether the origin is within or outside the bone. The red tumours are generally not malignant, but they may have in them other ingredients of a malignant nature. Thus, you see in this specimen a large tumour growing from the fibula, composed of a soft matter and bone; the former consists of a structure which can be called by no other name than medullary sarcoma, while in parts there are nodules of the red myeloid matter. The lad from whom it came died some months after amputation, with the same disease in the lungs and spine, here also composed of the same three elements.

All endosteal sarcomas are not of this "myeloid" form, however. The latter, as we have said, grow in the end of the bone chiefly. Those that grow in the middle of the shaft of a long bone are generally harder and have less tendency to distend the bone; they rather erode it and thus weaken it, so that it may break and the growth sprout from the fracture, as happens more often in some cases of carcinoma. Similar firm fibro-sarcomata occasionally grow in the body of the lower or upper jaw.

Occasionally, as in this instance upon the tibia, melano-sarcoma is found affecting the bones secondary to such melanosis elsewhere.

Myxoma.—The gelatinous forms of sarcoma, such as contain mucin in the tissue, are not infrequent in the bones. They often present the characteristic structure of "mucous tissue" or myxoma (see Plate III), but frequently they graduate into the consistence of soft cartilage (myxo-chondroma), or they graduate into the consistence of firm sarcomas. When well characterised, they form soft tumours of almost gelatinous consistence and glutinous feel on section. Their substance may be nearly pure, or permeated throughout by threads or networks of bone running through them. They are generally endosteal in their origin, and are affiliated by Virchow to the marrow, which approaches in its developmental stages very closely to mucous tissue.

Angioma, aneurismal tumours of bone.—Much interest attaches to the vascular tumours of bone which often assume great importance. A great deal of obscurity still beclouds their pathology, a large number of cases are recorded, and they have been collected and considered by several authorities in the endeavour to reduce them to some clear and simple pathology. Two views have competed in regard of them—one which regards them as essentially aneurismal, being primary dilatations of the vessels; the other, which considers them to be solid tumours softened down to blood-cysts, or permeated by wide vessels. In favour of this latter view is the frequent presence of small dilated vessels in the outer wall of the main cyst, and the gradation of these pulsating tumours of bone into such examples of osteo-carcinoma and osteo-sarcoma as are highly pulsatile. In favour of the former view that regards them as aneurisms is the fact that some have been cured by ligature of the supplying vessel. The vascular tumours are very rare, and it is not easy to examine them minutely. They are generally nævoid, being composed of dilated intercommunicating channels, separated by fibrous and osteo-fibrous plates and beams, which form their boundaries. The lining of the blood channels being very thin, and like that of the veins in structure, sometimes the channels are large, sometimes they are smaller. Often the search for the materials of actively-growing tumours has not succeeded in discovering in some of them anything which could be regarded as a matrix whose blood-vessels were dilated. They sometimes have clinically a very deceptive resemblance to aneurism. Thus, when such a tumour arises in the inner end of the clavicle, it may be a most difficult matter to distinguish the case from one of subclavian aneurism thrusting forward and expanding the bones. The differences that have been noticed in examples of pulsating tumours of bone which we have carefully examined have led us to believe them to be of different natures. Some appear to be truly dilated vessels which are in direct connexion with the arterial system, and hence receive the arterial impulse and are pulsatile. Others have a more obscure origin, and appear to be formed by cystic changes in vascular sarcomatous tumours arising in the medulla of the bone; such are those which are formed especially about the knee-joint, where the head of the tibia for instance may be found greatly dilated by a tumour of blood which, when carefully searched, reveals nothing more than blood and blood-clot. It is not, however, only in bones that this difficulty in explanation of blood-cysts is met with; we shall have to draw your attention to like instances of obscure blood-cysts in the soft parts, especially the deep parts of the limbs where such blood-clot-like tumours often have a very grave significance from their tendency to prove malignant.

Carcinoma.—The rapidly growing forms of sarcoma often prove very malignant by returning after removal and by metastasis to other parts. These characters of malignancy are of so much greater importance than any mere histological distinctions among tumours that they have always received the chief attention, and have hitherto in practice almost entirely overpowered the attempt to distinguish accurately the carcinomas, *i. e.*, tumours composed of an alveolar framework containing epithelioid cells, from the sarcomas whose composition is a homogeneous mass of cells connected by intermediate "intercellular" tissue more or less fibrillar.

Indeed, authorities are just now perhaps farther off than ever from a clear understanding as to the mutual limits of these two classes of tumours in bone, especially since Billroth has proposed to regard some of the tumours of alveolar structure as alveolar sarcoma rather than as carcinoma, on account of the want of resemblance of their cells to epithelium. We believe that this is a true view as to many cases. Thus, in a great tumour of the base of the skull which we recently examined, and which presented on section a minutely reticular structure to the naked eye, we found a very well-developed alveolar framework, and yet the areolæ were filled with cells which were round, and were separated from each other by an intercellular substance. This appearance of areolæ enclosing round sarcoma-cells is often caused by the section having cut bundles of spindle-cells which were running in different directions, some being divided across, while their neighbours around were cut lengthways (Plate II, *c*).

A long time and a great number of observations will be required to settle this question; in the meanwhile it is certain that tumours of a true carcinomatous structure are found in the bones. The most certain examples are those in which several secondary cancerous tumours are found in the skeleton in cases of carcinoma of the breast, or, more rarely, of the uterus. The nature of the primary cancer determines the nature of the secondary. This is a point of great interest in the history of cancer, and one you should bear in mind as of practical importance, that, various as the kinds of malignant tumour are, yet the several growths found in any particular case of multiple malignant tumour are all of the same kind. The tumours secondary to scirrhous carcinoma of the breast are found to be themselves also scirrhous carcinoma. They may differ a little in their consistence from the primary tumours, the secondary growths being rather softer and more rapid; but the structure has the same histological character.

Besides such secondary carcinomas of the skeleton we find some that are primary. Thus, we recently examined several cases of true carcinoma arising in the bones of the skull. In one of these the basilar bone was the chief seat of the disease, which had extended to the jugular foramen, and destroyed the pneumogastric and spinal accessory nerves; it was very interesting to observe that in this case the trapezius and sterno-mastoid muscles had wasted away almost completely, notwithstanding that they possess an independent spinal nerve-supply. But most of the cancerous tumours of the skull occur in its vault, in the form of more or less and, perhaps, remarkably numerous patches of growth that often appear to arise in the diploë, and to grow at the expense of the bone until they thrust themselves through one side (generally the inner) or both sides, so as to push in the dura mater and lift the scalp. When the calvaria is torn off in the usual process of opening the skull such tumours are often left attached to the dura mater, and so appear to belong to it rather than to the bone; hence it is often not easy to say which tissue they really arose in, especially as they generally begin upon the inner face of the calvaria, and lie at first between the bone and dura mater. However, their origin from the bone is generally shown by their being covered by a complete layer of the dura mater, while the bone is hollowed out by their growth. In the dry bone such hollowed spots may appear as clearly punched holes, or they may be filled up with a network of bone belonging to the growth; the edges are abrupt and show either no diseased change or else a growth of bony papillæ around the hole. These growths of papillæ often form a quite striking feature in the disease; it is interesting to notice that they are in no degree cancerous, they embody the effect of the active flux of blood, and consequent extra supply which the cancer has induced in its own neighbourhood. In these cases of cancer of the cranium you will rather frequently find that the spinal column has similar patches of cancer, and, in some cases, the tumours are scattered through the pelvic bones, ribs, sternum, &c.

One example of such disseminated cancer of the cranium and other parts of the skeleton proved to be of an exquisite *colloid* structure. It was shown by us at the Path. Society, 1872. We have also found medullary sarcoma scattered in the skeleton with the same distribution, so that such a dissemination is by no means peculiar to carcinoma.

Carcinoma is comparatively little liable to undergo ossification; nevertheless, we sometimes find the larger trabeculæ of the alveolar framework of the tumours partly changed to bone—osteo-carcinoma.

Besides the origin of tumours within the bones primarily or secondarily in the cases just described, the bones are also liable to be reached and attacked in the spread of carcinoma, and in this way we meet with epithelial cancer running in the bones. Thus, in this example from a patient of Mr. Cock the upper jaw is largely implicated in an epithelial cancer of the cheek, and it is far from infrequent to find a cancerous sore on the leg reaching the tibia, invading it and extending in its structure. The epithelioid cells of the cancer, as they grow, insinuate themselves into the Haversian spaces, and the bone slowly disappears before their growth, so that in a section taken from the edge of a cancer where growing into a bone you see the remains of the bone reduced to mere spicules which remain embedded in the new epithelial cells of the cancer; these spicules you may see reduced to the minutest fragments, and it is a most tempting pursuit to find out whether the proper cells of the bone do or do not give rise to the cancer cells. We have spent many hours in the endeavour to decide this question, but without adequate success. The spicules of bone left embedded in growing cancer commonly show those excavations of their edges which are called Howship's lacunæ. These are found packed full of cancer cells; but whether by multiplication of the bone cells we could not assure ourselves. The examination of the growing edge of a tumour in bone, to ascertain the behaviour of its cells, is very difficult. Sometimes the border of the growth shows a great quantity of very small cells, such as are called by Virchow granulation tissue, and these are described as arising by multiplication of the bone-cells. Analogy with the behaviour of the cells of fibrous tissue under similar circumstances would support this view strongly; but we have not been able to convince ourselves of its actual occurrence.

For *osteoid cancer* see *osteoid chondroma* (p. 54).

Osteolytic cancer.—Some examples of cancer in bone are very far from appearing as tumours; they rather give the appearance of local osteomalakia. The interior of the bone at the affected spots contains soft reddish matter in the form of a rounded patch; there may be openings through the superficial hard layer of the bone leading into this patch. The red substance looks like injected marrow. It is composed of cells which are small and irregular in form and contains one or more small nuclei. We have seen this form of disease distributed extensively in the skeleton, especially in the calvaria. Such cases have been brought before the Pathological Society, and are found scattered through the journals. A very characteristic case is given by Leubuscher. Such formations appear to be of the same nature as those called osteolysis by Lobstein; such soft cancer of bones is very apt to be widely distributed, and then is found in the form of rounded patches in the vault of the skull, in the pelvis, sternum, vertebræ, and long bones, having indeed just the same distribution as we have already described as belonging to multiple carcinoma and colloid.

When attacking the bodies of the vertebræ it should be noticed that cancer has the very peculiar effect of shortening the spine, often remarkably, so that the stature of the person is reduced considerably. The new growth occurs in the substance of the vertebra, and, being soft, is squeezed out by the weight it has to bear, so that several of the inter-vertebral substances meet together, the cancer being forced out to occupy a position around them. Sometimes cancer grows in the laminæ and reaches the nerves, giving rise to a very painful form of paraplegia. Such diffused malignant disease of the bones may cause slow, painful, wasting cachexia. Its presence should be considered among the probabilities in obscure cases with such symptoms.

Cystoid disease of the bones.—A few cases are on record, as by Froriep and by Engel, of cystoid change distributed through the skeleton, the bones wasting into cystic cavities so that numerous fractures occur. The cysts begin as red spots, the cystic change follows. When small they have no wall, afterwards they grow greater and become multilocular, lined with connective-tissue membrane, and full of a dark fluid or a pappy material. There is some obscurity about the nature of these cases. They would appear to be allied to those cases of osteolytic cancer we have just alluded to. But they still more resemble osteo-malakia, to which both in clinical features and pathological products osteolytic cancer bears so deceptive a resemblance.

Cephalhæmatoma is the name given to those blood tumours which occasionally arise on the heads of children at time of birth. We do not allude to the scalp tumours, which are more common, but to effusions of blood which take place beneath the periosteum, and which is a rarer affection. The blood may be absorbed, but sometimes it breaks out externally, leading to a slough; or it may involve the bone and penetrate to the brain, leading to death.

This last affection leads us to remark that in *scurvy* the blood is poured out, often in large quantities, beneath the periosteum, and that this subsequently hardens, and even becomes vascular; thus Busk has succeeded in injecting such a mass from the vessels of the bone.

Hydatid in bone is very rare, but it is occasionally met with, as in this preparation of tibia, in the head of which is an encysted hydatid containing many daughter hydatids. This is a unilocular hydatid, the whole being contained within one parent cyst. Here, also, are two preparations of hydatid of the spinal column. Both of them are instances of multilocular hydatid. There are very numerous hydatids, many very small, extending widely in all the tissues about the spine under the pleura, and also perforating the spine and destroying the vertebræ, raising then the dura mater as a tumour which compressed the spinal cord and caused paraplegia which was fatal through sloughing of the bladder. In these multilocular hydatids there is no common enclosing parent, but the cysts are found budding one from the exterior of another, producing often short chains of three linked together by the yet undivided bud-stalk. Microscopically the tissue of the hydatid, &c., has the characteristic concentric lamination, but there are no hooklets, in other words, the sacs are acephalous. Although hydatid of the spine is a rare disease, yet as many as ten cases may be collected. In this case, which came under our observation in 1871, the duration of the symptoms was eleven months.

Tubercle in bone is still, as always, a theme of contention among pathologists. Some consider as tubercle all the numerous cases in which we find a caseous slowly progressing suppuration tending to the softening down of the proper tissue within the bone. This we have already described under the title of caries. There is much good reason to consider that such caries is of the same nature as local tuberculous disease. In either case the proper tissue is destroyed by a lymphoid formation, which perishes, itself and the proper texture dying together. In either case the disease is slow and tenaciously obstinate. Any one of the whole class of tuberculous diseases, including caries itself, is very often and very significantly associated with others of that class, so that by their concurrence as well as by their analogies and identity of character these diseases form one great class. Thus the scrofulous or tubercular caries of the joints in particular is very frequently associated with pulmonary phthisis and other tuberculous diseases, as of the intestine, larynx, &c. There is then, we think, sufficient ground to class the caseous disease of the bones with the caseous disease of the glands, spleen, supra-renal capsules, kidney, testis, lungs, &c., and if this be agreed it is comparatively unimportant by what name you may call the group which has common features of such main importance, especially when there are no distinctions among them to be in any way compared with their common features.

On the other hand, if we limit the meaning of the word tubercle to the small round knot known as "miliary tubercle" or "grey granulation" in the lung or on the serous membranes, then tubercle in bone becomes apparently very rare, and certainly unimportant, as distinct from caseous caries,—unimportant because either its course is identical with the caseous caries in slowly breaking down the bone-tissue into a caseous ulcer, or else it is only a small part of a general tuberculosis; and apparently rare, perhaps because it is so comparatively rarely searched for in those cases where it is most likely to be present, namely, cases of death by general tubercle. In these cases, if the medulla of a bone, as the end of the femur or the patella, be examined, there will often be found a few tubercles scattered through the bone. Practically, then, tubercle in bone is either the same as caseous caries or it shrinks to the dimensions of a pathological curiosity.

DISEASES OF THE JOINTS

We shall only briefly run through this section, as the whole subject is given in the surgical lectures. The joints are composed of the articular extremities of the bone, which are cancellous, and covered by a more compact layer of osseous tissue; this, again, has over it the articular cartilage, and the two ends are connected by a capsular ligament, lined by a synovial membrane. Opinions vary as to whether this membrane terminates at the end of the cartilage or is carried over it; but we believe, in the adult, it ceases at the cartilage, the cells which are seen on the latter being its own superficial layer, and not those of a distinct membrane. The synovial membrane consists of a delicate vascular tissue, covered with epithelium, and from the surface of which there hang down a number of vascular fringes, which become enlarged under certain morbid conditions. The diseases of the joints are nearly all either inflammatory or degenerative, or a combination of these.

Inflammation.—*Simple and traumatic arthritis.*—For the sake of classification, the distinctions into the various forms of inflammation, and into acute and chronic, are made; but in particular cases it is often difficult thus to distinguish. The changes which go on in the synovial membrane are very similar to those witnessed in other serous surfaces—an acute process accompanied by effusion of lymph or pus; or a more chronic process accompanied by a simple serous effusion, or by the production of more solid material. *Simple synovitis* is seen best after an injury where the ordinary symptoms and signs of inflammation exist, with swelling and effusion into the joint, and which may subside again in a few days. If the joint can be inspected at this time, there will be found great vascularity of the membrane, with increased serous effusion, and, probably, some flakes of lymph; the latter will be seen covering the synovial membrane, but not the cartilage, except in young subjects, where we have been able to detect a distinct layer of lymph, the part beneath being highly vascular. When the injury is severe, suppuration occurs, the joint becoming distended with pus. This result, which is always very dangerous, is, unfortunately, too often excited by what appears to be a disproportionately small degree of damage, as when a violent and perhaps fatal suppuration of the knee-joint arises from a careful attempt to remove a loose cartilage, or a fragment of bone from it; at other times the severity of the arthritis is only in accordance with the character of its cause, as when a neighbouring abscess breaks into the joint. In any such circumstance the joint is found deep red or purple within, and containing a quantity of pus, generally of glairy consistence from some admixture with synovia.

Should a case of traumatic arthritis be protracted for several weeks before it comes under your examination, the cartilage will be found attacked, and at last the bone will become affected. Of course, in some cases the bone is implicated from the first, as in compound fractures into the joint. The mode in which the cartilage is removed differs, we think, in the appearances it presents from the process of its removal in scrofulous joint disease. At least, we have nearly always found in grave traumatic inflammation of the joints sound-looking cartilage standing in greater or smaller patches like islets with abruptly defined margins. These islets are firmly rooted on the bone beneath, and appear to waste away at their edges only; while in scrofulous inflammation the cartilage is found to soften down, and be removed in thin flakes, coming soonest from the surfaces of the bone at the points where they are naturally in close contact.

Most arthritis that is not due to mechanical injury or other obvious cause is reckoned rheumatic, but generally many joints are affected in rheumatism. Trousseau, however, speaks of rheumatic inflammation limited to a single joint, and we sometimes see monarthritis accompanied unmistakably by the conditions of rheumatism; but we equally undoubtedly do meet with simple arthritis of a rather acute and severe character where there is no sign of any rheumatic state about the patient; such a case is just now in Petersham ward, and Mr. Davies Colley tells us they are far from infrequent among the surgical outpatients.

Simple effusion.—As in the pleura we may meet with a chronic effusion of serum, without any attendant symptoms, dependent on a morbid (though unappreciable) state of the serous membrane, so a similar process may occur in a joint, whereby a large secretion of fluid and swelling of the joint take place, producing the affection known as *hydrops articuli.*

Pulpy degeneration.—A still more chronic process, of a different character, whereby a solid material is formed, and the membrane altered in character, constitutes the pulpy degeneration of Brodie. In speaking of the periosteum, we said that when an exudation occurs from a membrane and into it, the membrane itself becomes altered and incorporated with the new material; and here, too, by a slow production of lymph from the synovial sac, it becomes swollen, soft, and so changed in form, that from being a thin tissue it becomes a soft and thick one, and then, as the process still continues, it grows thicker and thicker, until this pulpy membrane is produced. We use the word "grows" advisedly, for when the synovial membrane has become vascular, and it is seen to be gradually increasing in thickness, the process is more allied to a growth than to organisation of previously effused lymph. The joint becomes much enlarged and soft, and the disease can be easily detected by the *tactus eruditus* of the surgeon. Although the affection in its simplicity may be looked upon as a special form of disease, we believe lesser conditions may be constantly met with in various morbid states of the joint. Thus in severe cases of general joint disease involving the cartilage, the synovial membrane may be found converted into a similar pulpy gelatinous state. Indeed, in our experience it is rare to find the pulpy condition we have described, unless characters of a scrofulous arthritis are present in the joint.

Another chronic diseased condition of the synovial membrane is where a number of vascular fringes hang down into the joint; these appear to be enlargements of the natural fringes described by Rainey. If you look at these various specimens of disease of the knee-joint, you will observe this condition, and so also in some others to which we have already alluded.

Rheumatic Inflammation.—It is fortunately not often that patients die of acute rheumatism, so that anatomical examination of the joints in that disease is as rare as clinical observation of them is frequent. When an opportunity is offered you are sometimes struck with the slightness of the change within the joint; it is scarcely noticeable; indeed, it would probably not be recognised unless a minute examination were made; the only appearance observed being a slight increase of fluid, the hyperæmic condition of the synovial membrane, which, no doubt, had existed during life, having now disappeared. If you remember how fleeting is the pain and swelling of the joints, that a part affected acutely at one moment may in a few hours be quite well, you will perceive a reason why no structural change would be manifested.

But all cases are not of this slight degree; sometimes you find the joint much distended with turbid fluid, and its surface pink from congestion of its vessels. In other cases large flakes of lymph float in the fluid, or a coating of lymph is found lining the whole of the synovial surface of the joint. In these specimens you see large masses of lymph which were removed from the knee-joint in cases of acute rheumatism. Again, it is not only within the joints that you see evidence of inflammatory action in rheumatism, but also in the *sheaths of the tendons*; we have several times found the sheaths of the extensors, where passing the wrists, full of the same turbid flakey fluid as was present in the wrist-joints.

This considerable alteration in acute rheumatism enables you to understand how it is that recovery is often so slow and incomplete. It is true that the behaviour of the joints after acute rheumatism is usually in contrast with the behaviour of the heart when it has been affected—a contrast that is sad enough on the part of the heart, which so frequently remains permanently affected, while the joints entirely recover their suppleness and freedom (we shall see the reason of this when treating of rheumatic heart disease), but, although rarely, rheumatic joint disease does leave severe after effects. The ligaments contract so much that the joint is permanently stiff; or the synovial membrane is so damaged by inflammation that it will not reabsorb the fluid effusion, which consequently remains as a long-standing hydrarthrosis; or even, in some cases, an adhesion forms between the articular surfaces by bands of fibrous tissue.

There is another set of conditions connected with rheumatism in which a yet more intense joint disease may be sometimes observed. Thus the cardiac inflammation of a rheumatism may become ulcerative, and at the same time the joint disease may put on a destructive severity, the cartilage being removed, and a state of simple caries or ulceration of the bone established. This bone (you may be excused for not recognising it) is the trapezium from the left wrist of a man who was admitted under Mr Birkett for a chronic disease of the wrist-joint, following out of an acute rheumatism suffered some months before. The man had ulcerative disease of the aortic valves, and after his death the wrist-joint was found undergoing disorganising inflammation, including removal of cartilage from, and ulceration of, the bone. The connection of this with rheumatism is less direct because there is the ulcerative disease of the heart to consider, the result of which is to produce a general pyæmic tendency, but the joint disease relatively often in these cases leads directly out of an attack of rheumatic fever.

The post-mortem examination of simple chronic rheumatism is meagre in its results; such cases you find in women especially, more or less distorting the fingers, and often called by the name "rheumatic gout," which usefully covers a doubt its employer cannot solve. The amount of distortion would lead you to expect considerable changes within the joint, whereas you do not generally find the bony surface at all altered; the synovial membrane looks healthy. The only discoverable change as cause of the distortion is thickening and contraction of the ligaments, but you know it is not easy to see where ligaments are a little thick and contracted. The swelling you see around such joints in the wards is the result of œdema from one of those frequently recurring inflammations which make these cases so intractable.

Pyæmic and Gonorrhœal Inflammation, &c.—The most severe forms of inflammation of the joints are seen in connection with pyæmia. In this disease a dirty thin pus may be found in the joint after it has been affected for only two or three days; but as yet there is no destruction of tissue, the synovial membrane is only found somewhat more vascular; if, however, the patient live, a destruction of the joint would very rapidly ensue. This we have often the opportunity of seeing by comparison of the joints that have been surgically opened, with those that have been left to their own course, through the surgeon's despair of relieving by further interference. Thus, in a case we had not long ago, the right shoulder was attacked after the left. The left, which was first attacked, had been opened, but the right, which swelled when the patient's case was thought to be obviously beyond relief, was left unopened. The contrast of the states of these joints was very instructive. The left shoulder-joint, which had been longest diseased, but had been incised in time, showed no disorder beyond the confines of the joint itself. But the right showed a most frightful destruction, indeed, the scapula lay bare in a bath of foul pus. Such instances, not always so striking, we occasionally see, proving the importance of early giving free exit to the pus from within the joint.

Attention must be directed to the relations which the polyarthritis of rheumatism has to the polyarthritis of pyæmia. In both diseases the heart is apt to be affected. There is reason to think that the polyarthritic variety of pyæmia is a disease that differs from that variety which especially affects the lungs, liver, &c., and is a disease more amenable to treatment. In speaking of acute necrosis, we pointed out a curious variety of pyæmia which too often accompanies it, affecting the heart and kidneys in its secondary suppurations almost always, while the joints generally escape.

In speaking of *gonorrhœal* rheumatism, we should first note that in two cases which have occurred at Guy's, an obscure fatal pyæmia proved to have for its cause a gonorrhœal inflammation of the urethra, the evidence of this being the discovery of purulent inflammation of the prostatic plexus of veins, while the urethra was full of pus. *Gonorrhœal* rheumatism is usually a subacute form of a pyæmic inflammation. We mention it here in connection with pyæmia, not because pus is formed, for this we think is very rare, but because we think it owns a similar cause. It is only fair to state to you that opinions vary much as to the existence of this form of disease; but our own experience leads us to the conclusion of its frequency, and this is supported by the fact, that we believe similar affections of the joints may be witnessed in women and other persons who are suffering from purulent discharges. Gonorrhœal rheumatism is characterised by a subacute inflammation, affecting sometimes the knee and larger joints, but more especially the wrist and ankle, more commonly the latter; and when we speak of this, we mean not only the true ankle-joints, but all the smaller joints of tarsus in the neighbourhood; these deep-seated joints, with the ligaments, become affected, and a very chronic painful disease is the result, productive of a thickening and induration of all the articular tissues.

Along with gonorrhœal rheumatism you should consider *scarlatinal* rheumatism, in many aspects so analogous. Scarlatinal rheumatism is often nothing less than severe pyæmia, and evidently traceable to purulent infection from the sores in the throat. But all scarlatinal rheumatism is not of this grave kind; you not infrequently meet with little children recovering from moderately severe attacks of scarlatina, during which no loss of tissue had occurred in the throat, but they complain of their joints, which become red, and swell; afterwards the swelling goes down, and no evil comes of it. Also it will be remembered here that these rheumatic sequelæ of scarlatina sometimes induce, among the other local disturbances, acute endocarditis.

Puerperal rheumatism stands in a very similar relation to pyæmia, but suppuration is relatively more frequent. The same is true of that following *smallpox*. Besides these, we occasionally meet with a class of obscure cases, either in the wards or on the *post-mortem* table, in which polyarthritis of more or less grave kind is associated with pellicular inflammation of the fauces, and it is difficult to say whether the disease is to be regarded as diphtheria with rheumatism related to it like it is to scarlatina, or as rheumatism with pellicular pharyngitis. In one such case, two or three years ago, in the clinical ward, the tongue became much swollen, and on one side of it there formed a considerable slough; yet, after much danger of suffocation and severe articular inflammation, which did not reach suppuration, the patient, a stout, flabby, middle-aged man, made good his recovery.

In short, the connection of polyarthritis with febrile diseases is a very interesting subject, and would well repay more special inquiry than it has yet received. It may be generally said that any of the fevers may be accompanied, or more generally followed, by rheumatoid polyarthritis. The occurrence is rare in the case of some of the fevers. But with others it is so frequent that it becomes important to be aware of it. Thus, some epidemics of *dysentery* have been marked by the frequent occurrence of rheumatism as a sequela of the intestinal disorder in the subjects of the attack. This connection of dysentery with rheumatism was observed very long ago, and it has more recently drawn attention, as in the Norwegian epidemic a few years ago, by Homan and Harting, and in still more recent instances. In some of these cases endocarditis accompanied the rheumatic disorder.

Scrofulous Inflammation, or White Swelling.—The remarks we have already made in reference to tubercle in bone will prepare you for the difficulty of knowing what is exactly understood by scrofulous affection of joints. The old opinion was that tubercle formed in the bone, and then softened, and set up mischief in the neighbouring joint. There is no proof, however, that this is generally the case, but rather a synovitis of a slow nature occurs in scrofulous children as in adults, and the articular ends of the bones become involved in the inflammation. It is not often that we meet with a chance of examining scrofulous disease of the joints in its earlier stages. In January, 1870, the knee-joint of an amputated limb, which had been removed for caries of the head of the tibia, showed in its synovial membrane tuberculous ulceration in a most interesting way. Lenticular ulcers, of a size from millet seed to hemp seed, being distributed closely and regularly over the synovial membranes, which otherwise was little altered. The joint was only secondarily implicated in the disease. This, however, although it suggests, yet would not prove such tubercles to be present when the knee-joint is the primary seat of the disease, for we shall often have to mention to you the frequent formation of tubercles close about old caseous disease, when there is no opportunity of proving this caseous disease to be itself tuberculous; and you will find that several conflicting interpretations are put upon this association, which interpretations we shall allude to on a more convenient occasion. Similar cases of tubercle in the synovial membrane have been recorded by other observers. In reasoning from the rarity of their occurrence, we must remember how unusual it is for the early stage of joint disease known to be scrofulous to come under anatomical examination. But the later stages of the disease are amongst the most frequent objects of our morbid anatomy. In these, if extreme, we find the soft parts of the joint diseased excessively; the interior of the joint being brought to a state resembling that of the sinuses which communicate between its cavity and the outside of the limb, that is, like the interior of very old abscesses. The synovial membrane, or rather what was the synovial membrane, shows degrees of that pulpy degeneration which we have before described, but it is deep purple or red on its surface for the most part, and may be in many parts eroded. The tissue outside the joint is more or less consolidated into a white fibrous mass, soaked with serum, and showing the remains of fat and muscle, &c., when looked at closely. The tissue is in places charged with inflammatory products which may break down, forming abscesses that burst into the joint, or burst upon the surface of the limb, so causing *sinuses unconnected with the joint.* The cartilage is in such old cases nearly all destroyed; the destruction shows itself, first, where the bones naturally press against each other in the actions of the joint; the cartilage generally is gradually thinned away there, and when reduced much may be found as a thin pulpy layer, separating as a flake from the surface of the bone.

But sometimes when the joint is examined at an earlier stage, the cartilage is found parting from the bone, as though it were shed like cuticle, or in slower cases the medulla of the bone sends up vascular granulations that pierce the cartilage in many parts, so that it looks porous, meantime softening down while the granulations project in the joint, and meeting with similar granulations may unite and ossify, producing ankylosis. In these cases the bone is observed charged with red lymph, and then the inflammation of the bone is to be regarded as the main disease which implicates the joint through contiguity. This, as we have said, was thought by some surgeons to be the case universally. Tuberculous inflammation of the bone softening down was supposed by them to be always the starting-point of a mischief that afterwards implicated the joint, but certainly it is not so in all nor even in the greater number of cases. For those examples, wherein the disease has not gone on to the utter demolition of the components of the joint, and where a considerable amount of cartilage is left still suffering the destructive processes, show good evidence of the cartilage being partly taken away off the free surface. Thus, we have often seen examples of the fact noticed by Sir Everard Home, where the cartilage on the borders of the faces of the joint are grooved by the fringes of swollen synovial membrane, which project between the opposed cartilaginous faces; without giving unqualified support to the opinion that these synovial fringes absorb away the cartilage, it is clear that we must consider the removal of it to be so far effected by a process belonging to the inside of the joint, and not to the bone beneath. Probably the pressure of the fringes, swollen and inflamed as they are, causes the disintegration of the parts subjected to it, while the inflamed state of the cartilage itself favours the change. This is rendered very likely through a fact we have already mentioned, namely, that the cartilage goes most extensively and decisively off the parts of the joint's surface which are most firmly pressed together, such as the convexities of the condyles of the femur, and the opposed parts of the tibia, while it lasts very long on the facets of the patella and the other portions of surface which are little compressed; we hence see how pressure will take the cartilage away. As further proving that the disease begins within the joint, you find the bone around, in many cases, to be simply softened, pale, and very green in texture. Its state, in short, is exactly that we have described as found in the muscles, &c., outside the joint, a state, that is, of wasting away from disuse, so that it is charged with the watery, fat, and œdematous fluids that generally surround the stagnant neighbourhood of old weak inflammations. But such cases as affect the bone chiefly and primarily, show caries, and perhaps necrosis of its articular ends, which are eroded and generally dense, with evidences of active repair showing themselves in a quantity of spicules of new bone, that project in sharp points around the ends of the bones, following the course of the ligaments which they generally are formed in; or the articular faces will be covered more or less completely with new bony matter, tending to repair the mischief by ankylosis.

Chronic Rheumatic Arthritis is a name which the authority of the most careful and laborious describer of the disease (Dr Adams, of Dublin) has rendered classical. This name sufficiently well denotes the main characters of the disorder. It had been called *nodosity of the joints* by Haygarth, which was not an unsuitable name. When affecting particular joints it had been known by names whose application is either partial or not distinctive, such as *malum coxæ senile*, which would wrongly infer that the disease does not attack young people, or *arthrite seche*, which would not concur well with the fact that at some periods of the disease there is often great effusion of fluid into the joint, or *arthritis deformans*, but deformity of the joints is produced at least by gout also. Cruveilhier, who appears to have been the first to claim for this disease a clinical recognition, proposed to call it *usure des cartilages articulaires*. But this name puts forward the erosion of cartilage much beyond its true importance in the disorder. Such erosion is common in advancing life, when there is no trace or suspicion of the disease in question. However, as we have said, the name chronic rheumatic or rheumatoid arthritis is now universally accepted for this disorder, when recognised by its proper anatomical characters; though during life we often hear used for it the vague name "rheumatic gout," which serves as a cover over its employer's doubt of the pathology of his case. Sir B. Brodie introduced gouty deposit as a feature of its anatomy, but when such deposit is found it must be regarded as a complication.

The disease has more than a full share of the vagueness of definition of rheumatic disorder, for the chronic and obscure cases of rheumatism are those that especially tend to fall into it. Very often there is no reason to consider the case to be rheumatic, as when it arises from a sprain or concussion to a single joint. But in many cases it has followed out of rheumatic fever in such a way as to appear to be a chronic continuation of that disease, just as chronic pleurisy is a continuation of acute pleurisy. Some choose to interpret this so that the rheumatic fever is held to have been not true rheumatic fever, but "acute rheumatoid arthritis." This is, however, entirely arbitrary. As the cause of this disease is obscure, so also its earlier stages are not well defined; it is only in the later development of its effects on joints that it becomes clearly recognisable. The joint is sometimes described as dry in the first stage, but this statement is, we think, made on account of the dry grating feel which the eroded cartilages of elderly people impart to the hand during movements of the joint. This erosion, however, does not constitute rheumatic arthritis. In its earlier stages the joint shows only the condition we have already considered as *hydrops articuli.* The cavity has in it an excess of clear synovial fluid, while the wall of the joint is more or less thick, and its flocculent processes are increased in number and size, and more or less deeply red from congestion. It is thought that by this effusion the ligaments are stretched and lengthened so as to favour the partial dislocations which sometimes occur, but the dislocations follow upon such changes in the ends of the bones as sufficiently explain their displacements.

It is at a later stage that the peculiar characters of the disease show themselves. These affect chiefly the bone and its cartilaginous covering. When the disease is characteristically established, the joint is seen to be much deformed; the most striking peculiarity being the production of a quantity of new bone in nodular masses around the edge of the articulation, and occasionally of separate pieces in the capsular ligament. One may form an idea of the appearance of the joint in this advanced stage by supposing that its cartilage should be removed, and the ends of the bone made so soft that they partly rub down into a bony paste, which is squeezed out from between them to solidify in the form of a rough ring, turned over all around the edges of the worn and polished articular face. If, then, the capsular ligament be conceived to be thick, and the synovial membrane thickened and here and there flocculent and congested, or provided with polypoid, fatty, cartilaginous, or osseous growths, a very good idea of the superficial characters of the disease will be obtained. The neighbouring bursæ are generally implicated, and add to the deformity and disablement.

The behaviour of the articular cartilage in this disease has received the great share of attention, but we think its importance has been exaggerated. Thus, it is said that the disease really consists of an irritative proliferation of the cells of the cartilage, producing a softened and overgrown state of its substance. The softness then, it is said, induces a wasting of the parts subjected to friction and pressure, while the soft overgrowth of cartilage sprouts at the edges of the joint where it is not repressed, and bulges out in the form of an irregular rim, which subsequently ossifies, so the bony masses around the joint are accounted for. But the worn and eroded state of cartilage found in chronic rheumatic arthritis is very far from being proper to that disease; indeed, erosion of cartilage is very frequent. It may be said that after the thirty-fifth or fortieth year it is usual to find such erosion, though chronic rheumatic arthritis is a comparatively rare disease. It might even be fairly suggested that the constant presence of this erosion of cartilage in rheumatic arthritis is only a coincidence from both disorders belonging to the years of declining life. But the histological changes are the same, whether the erosion is with or without the changes in the other parts, and it would, therefore, seem very reasonable to think that the implication of the other structures of the joint comes about after the destruction of cartilage has progressed beyond a certain limit so as to throw the wear and tear upon the bony tissue unprotected by cartilage, and so arouse in it and its neighbourhood subinflammatory reaction, thus constituting rheumatic arthritis in its more obvious form. Nevertheless, though this view is plausible, we think a large number of observations induces the belief that the erosion of cartilage does not lead so simply on to pronounced arthritis, but rather there is some other factor which is required to set up the inflammation, such as rheumatic fever or a blow or sprain; and this latter factor is able to set up the rheumatic arthritis in the absence of degenerative erosion of cartilage, as is proved by the not very infrequent occurrence of that disease in the earlier years of life.

When the cartilage is removed the exposed bony surfaces polish each other by mere attrition; their faces being by this means compressed and condensed and the earthy matter is then brought to an even shining surface, bearing often a beautiful polish, as though glazed or enamelled. This layer of enamel, when examined microscopically, is confused and almost structureless, so that it evidently is not a new development, but is rather due to mechanical condensation of the old bone. By this rubbing down and condensation the ends of the bones are gradually worn away, so that, as in these specimens of hip-joint, you will see the head of the femur partially ground off, the ligamentum teres entirely removed, the articular face flattened and surrounded by a fringe of new osteophyte about its edge; also the rim of the acetabulum is partly worn away, so that the cavity is shallow, while it has a rough irregular rim of new bone around widening it; both surfaces are polished brightly where they have worked upon each other, thus supplying one quality of the departed cartilage — its smoothness—while the other—its resilience—is entirely wanting.

In this case, a longitudinal section of the neck of the femur, shows that the flattened mass of bone, which looks like the head of the bone deformed and pushed down on the shortened neck, is really new bone formed around the stump of the neck of the femur, the head being nearly all ground away. Thus, it would appear as if the trituration, which we just now spoke of in illustration, really had occurred, and no doubt the wearing away of the bone is such a simple mechanical process, but we do not think the new bone around the worn head is formed by anything bulging from the joint, as Rindfleisch's description appears to suppose, but rather, we regard the new bone as due to the chronic irritation of the periosteum and other fibrous tissues in continuity with the bone, such bone arising from the chronic inflammation ("ossifying periostitis") in the same way as spicules of new bone arise around the ends of bones in caries of the joints. But in chronic rheumatic arthritis, when compared with caries, the new bone differs characteristically. You will see by comparing specimens that in the rheumatic bone the new formation is in the shape of rounded nodules of dense consistence and close surface, while about the carious joint the new bone is in fine spicules and needles formed of open-textured bone. This difference is due to the inequality in the rate of the processes which produce the new bone, the slower product of rheumatic arthritis having time to undergo that condensation and smoothening of its surface, which ultimately occurs in all bone formations.

In the shoulder the disease has characters very similar to those seen in the hip, as you will observe in these specimens; the head of the humerus is quite altered, for instead of being round it is converted by new bone into a large irregular mass, having a polished oval surface on one side, where it meets the glenoid cavity; also, if much enlarged, it has another polished surface above, where it meets the under surface of the acromion; and sometimes even a third, to meet the clavicle. The glenoid cavity is expanded and flattened, but generally has not much new bone around it; probably the greater motion of the shoulder, as compared with the hip, prevents this; but in the capsular ligament there is some bony deposit; the under surface of the acromion is polished by the humerus, and this often is detached, so that it then is difficult to say whether it is the epiphysis separated, or a fresh development of bone; thus, as you see here, there is a distinct square portion of bone attached to the end of acromion by a ligamentous structure, and appearing exactly as if the point of this process had been broken off. We wish particularly to draw your attention to this, because only the other day this specimen was sent to us as one of fracture of the acromion, yet it exhibits nothing more than an example of chronic rheumatic arthritis. Two or three other similar specimens in the museum were formerly labelled as fractures. The cartilages are always destroyed, and the articular ends of the bones become polished or eburnated, and just as the ligamentum teres in the hip is altogether lost, so the long tendon of the biceps is often destroyed. As a consequence, the head of the humerus may become dislocated; then it is that examination of the joint, after death, may leave a doubt as to whether the luxation has been the result of disease, or whether an injury in the first place did not set up the latter. In many cases of supposed dislocation there can be no doubt that the disease is simply chronic rheumatic arthritis; but, at the same time, this affection may be caused by injury, as some of our specimens show; though it is doubtful whether the dislocation occurred at the first or was the result of subsequent changes.

In the *elbow* this disease is found less frequently than in the hip and shoulder-joints, but has the same characters. The cartilages are gone, the ends of the bones are polished, a quantity of new bone is seen surrounding the head of the radius and following the edges of the acromion and coronoid processes, as well as the front and back of the condyles of the humerus. In this specimen these elevated ridges could be felt during life, and much impeded the motion of the joint, suggesting ankylosis, which, however, never occurs in this disease. The capsular ligament also has some separate portions of bone in this case.

In these two specimens you will see in the fossa of the olecranon small bones or ossicles; but whether due to this form of disease we cannot say, as there seems no affection of the joint.

In the *knee* also the same changes occur; rims of new bone form along the edges of the condyles and around the head of the tibia. The deformed femur may grind away the head of the tibia on one side chiefly, as in this preparation, or otherwise wear it into curious shapes. The semilunar cartilages disappear, and the ligaments are so weakened that the knee-joint may become capable of many movements besides the two it is organized to go through; all being equally painful. The productive activity of chronic rheumatic arthritis is often more strongly manifested in this joint than in any of the others. It is not uncommon to find, besides the usual osteophytes on the bones, a number of large or small plates of bone in the capsule of the joint feeling like supernumerary patellæ. It is in this joint too that the growth of projecting and pendulous nodules from the synovial surface is most plentiful and free, as you might expect, remembering how large a share of papilliform projections and fringes it has within it in the alar ligament, &c. Such pendulous bodies may be either fatty or cartilaginous in structure or they may undergo ossification, and they sometimes separate from their original attachments, and are found as loose cartilages in the joints. This is an ordinary origin of such loose bodies, to which we shall presently advert more specially.

The other joints are also liable to suffer from this disease; the hands and fingers are often much distorted and disfigured by the nodulated stiffened and partly dislocated state of the metacarpal and phalangeal joints. In the *vertebræ* you may see, as in some of these specimens, a similar form of disease; new bone being produced along the edges of the bodies, which thus overlap each other, while the articular surfaces are expanded and polished. In these *pelves*, also, you will see a chronic affection of the joints resulting in a deposition of bone around the edges of the sacro-iliac and pubic articulations.

Chronic rheumatic arthritis generally does not lead to suppuration of the joint. But an exceptional case of disorganized knee-joint was exhibited at the Pathological Society by the late Mr Bruce, who had given great attention to the disease. In this case the joint had suppurated, although in its other conditions, especially from the quantity of nodular new bone around the articulation, it had the characters of chronic rheumatic arthritis. Sometimes, also, amputation of the thigh has been required to prevent the threatening supervention of suppuration of the knee-joint. Our experience concurs with that of Dr Adams when affirming that ankylosis is a very rare result of chronic rheumatic arthritis. Sir B. Brodie says ankylosis sometimes occurs, but the disease, as described by him, is not free from mixture with cases of a different kind.

Gouty Arthritis.—After a person has had but a few attacks of gout, the joint affected, usually the metacarpal joint of the great toe, shows a quite characteristic result in the presence of urate of soda in the cartilage. This shows itself as a patch of opaque white, like chalk; when this is small the cartilage may be but slightly affected in its nourishment by it; but if there is much of the chalk present the cartilage will be found thin and worn looking. Sometimes the chalky substance is found deposited in eroded cartilage, but then we think the erosion and the deposit are really independent though coincident. In severe cases the urate not only charges the cartilage but is found in the ends of the bones, as in this specimen from the great toe. Also it will accumulate among the ligaments and cellular tissue outside, forming the well-known "tophi" or pasty concretions, entangled in the meshes of areolar tissue, with which a little experience in the wards will make you familiar. It appears that when once this urate of soda is deposited in the cartilage it does not afterwards disappear, and thus it gives a very useful means of assuring ourselves of the former occurrence of gout when there is no history of it at the post-mortem examination.

The gouty condition of cartilage is sometimes found in the large joints. This preparation of knee-joint shows the whole surface, condyles, patella, and tibia, covered with white matter like mortar, which was proved by Dr Rees's analysis to be urate of soda. It would be well to notice here, however, that occasionally phosphatic paste occupies the interior of the joint, and has been mistaken for gouty deposit.

When the gouty cartilages are microscopically examined, its cells are found to be surrounded by needles of urate of soda, crowded around the cartilage cells so as to give them the appearance of mossy birds' nests. In the further stages the crystallization extends through the rest of the cartilage, and, as we have said, invades the bone and tissues outside, but it always begins in the cartilage of the joint. The joints appear liable to it very much in direct proportion to the severity of the mechanical wear and changes of temperature they are subjected to. The metacarpo-phalangeal joint of the great toe is by far most frequently affected; this suffers most among the joints of the foot in walking, as you may see by turning to look at the imprint of your naked foot on the sand. The influence of cold is probably instanced in the case of the external ear, in which you often find gouty concretions, so that in deciding on the gouty nature of a case you may often be much helped by finding these concretions in the auricle. They often arise without any pain.

It is not usual for a gouty joint to show the thickening of the ligaments and development of bone, &c., which characterise chronic rheumatic arthritis; indeed, in their complete development gouty and chronic rheumatic arthritis are very different from each other. But, as we have said, we sometimes meet the urate in cartilage which has undergone erosion, and we must remember that some consider erosion of cartilage to be proper to rheumatic arthritis, although we do not join in that opinion, but rather believe that erosion of cartilage belongs to elderly life just as deforming arteritis does. It certainly must be very rare to find gouty deposit complicating advanced and characteristic rheumatic arthritis, for we have never met with it. The diseases affect, on the whole, different sets of joints, rheumatic arthritis being best characterised in the greater joints and gout in the smaller.

Dislocation.—This is fully treated of in the surgical lectures, and therefore we will merely refer to our specimens, showing new sockets in *unreduced* luxations. Many of these show a surprisingly good substitute for the natural joint; a dense fibrous tissue takes the place of cartilage. The joint is made smooth by a new bursa, and surrounded by dense areolar tissue composing a fairly efficient capsular ligament. We may inform you that now and then you may meet with the affection known as *congenital* backward dislocation of the hip-joint. The child walks in a peculiar, stiff, upright manner, from the difficulty of balancing the trunk on the thighs, and has a rolling gait. On examination it is found that the head of the thigh bone constantly falls out of its socket. This is owing to a badly formed acetabulum, or to the absence of, or, perhaps, lengthening of, the ligamentum teres. It is possible that it may be due to a disease in fœtal life, as already seen in the sacro-iliac synchondrosis of the oblique pelvis; but of this there is no proof. You sometimes meet with persons who are constantly having a particular joint dislocated; this probably arises from injury in the first place. This head of a femur came from a woman who had had her thigh dislocated twenty-two times; the ligamentum teres is quite destroyed. Dislocation occurs from progressing *disease*. Thus, in the hip the head of the bone, or what remains of it, slips out of the socket on to the dorsum of the ilium. In the knee, when all the ligaments are destroyed, the bones are drawn out of their place and, like those of the hip, take a definite direction. The tibia is pulled outwards as though by the popliteus muscle, so that the outer condyle of the femur rests on the middle part of the head or on the inner tuberosity of the tibia, and the patella becomes attached to the front of the outer condyle. We would warn you to be careful in not confounding a simple dislocation and its supposed results with dislocation the consequence of disease, as has often been done in the case of the hip and shoulder.

Dislocation of cartilage sometimes occurs as a partial detachment of a semilunar cartilage in the knee-joint.

Ankylosis and Synostosis.—Ankylosis is a term which was formerly used as synonymous with stiff joint, originally implying that the joint was bent at an angle. Now, such unions in the joints are distinguished into two kinds, first, those dependent on a mere ligamentous union between the ends of the bone; and, secondly, those in which the union is effected by bone: the former class are called *ankylosis*, and the latter *synostosis*; what is called *false ankylosis* is mere immobility of a joint dependent on a stiffness of the ligaments and other parts external to it. Synostosis may generally be looked upon as a further stage of ankylosis, following upon a greater destruction of the joint; for if any cartilage be left when the disease ceases, or even if the articular lamella of the end of the bone be quite perfect, the union is merely by fibrous tissue, and in such cases some motion is still left. Where a good bony union is formed the disease has run such a course as we described in speaking of the severer inflammations of the joints, especially traumatic and strumous. The synovial membrane is replaced by a soft gelatinous matter, and at the same time the cartilage disappears, so that an examination of the joint shows the ends of the bones covered with granulations; and as the ends of the bones themselves have, no doubt, been participating in the change, a slight disintegration of the whole surface has taken place. From this the formation of vascular granulations has occurred; and now, if the active morbid process ceases, and the irritation descend to the degree compatible with formation of a new tissue, ossification takes place, and the two surfaces are united. In some cases of synostosis it is probable that the union has not been completed in this simple way, but that the original shafts having disappeared new bone has taken their place, and the part corresponding to the joint is formed from one piece. Thus, this specimen of ankylosis of the elbow does not show any traces of the original bones, but the single massive piece forming the angle seems altogether new. In other cases, however, you can clearly trace the outline of the original bone; in this one the head of the radius has remained free, while in these it is ankylosed in front of its natural position. In the knee both ligamentous and bony ankylosis may be met with, as also in the hip; in the latter you may often trace, as you see here, the irregular outline of the original diseased acetabulum fitted to the irregular process formed from the neck of the bone, the head having been lost. In the tarsal and carpal joints you will see by these several preparations that the most perfect bony union may occur. A remarkable disposition to ankylosis is seen in this preparation, showing the bones of the spine, pelvis, and lower limbs all united; there being an evident inclination throughout the body to a destruction of the joints, and a running together of the whole bony skeleton. Here is a remarkable specimen of a skull of a black man, showing ankylosis of the lower jaw.

Adventitious Growths and Loose Cartilages.—Actively growing tumours are not found in the joints. When such tumours affect the articular ends of bone the joints are generally preserved by the resistance of the articular cartilage. Only one example ('Virch. Arch.,' 644, p. 500) do we know of malignant tumour spreading along the synovial membrane; it occurred around the knee-joint. But we have already shown you that the joints in chronic rheumatic arthritis present pendulous bodies hanging into their interior, often in large number. This is especially frequent in the knee-joint, which is the favourite place for loose cartilages. A more local process of the same kind may cause the production of only one or two such bodies, which may then grow to a considerable size and become detached; thus it is that loose cartilages are probably formed, of which these are specimens. One, you see, is as large as a florin, while others are smaller. They are flat white, and smooth on the surface, although not even, having numerous depressions. Their structure is of fibro-cartilage, the cartilaginous element being in small proportion, or altogether absent in some; in others the structure is almost wholly cartilaginous, while in yet others there is some bone present; sometimes the apparent bone is only calcified cartilage, but in some examples there is true bone, with characteristic lacunæ and, perhaps, Haversian systems. The origin of these loose bodies has been the theme of much debate. It is generally agreed that the majority of them take their rise, as we just now said, from a localised process of the same nature as that observed in chronic rheumatic arthritis. But some arise as fragments separated from the ends of the bones. Thus Mr Poland removed from the knee-joint of a man who, in the dark, had struck his knee slightly against a corner of his bedstead, a piece of bone, that proved when the man died a few days after the operation to be a part of the patella; such an occurrence may easily be supposed to happen not infrequently. But we have more than once met in the knee-joint with grains of various sizes and of a consistence like soft boiled rice, resembling a good deal the "melon-seed" bodies of the sheaths of tendons. These appeared to be on the road to the formation of "loose cartilages," but much change was required to convert them into cartilage or bone.

Degenerative Changes in Cartilage.—*Degenerative erosion.*—Cartilage, which resembles the elastic coats of arteries in being evascular, also agrees with them in early showing a deficient power of endurance. A man has generally not reached his thirty-fifth year before his cartilages, like his aorta, show signs of being the worse for wear. The parts which reveal the change are those most pressed upon in use, such as the prominences of the condyles of the femur and the corresponding parts of the tibia; the patella has also a very special liability. A very good description of the anatomical characters of this change was given by the late Mr Alex. Bruce, and our experience fully corresponds with that on which he based his statements. To the naked eye the earliest sign of the change is a yellowish spot, which afterwards becomes soft and flocculent and wastes into a cavity. Sometimes it is more yellow, and sometimes more fibrous, and sometimes more excavated. These varieties of appearances are, however, usually successive stages of the change. The flocculent surface is best brought out in viewing the cartilage under water. Such a patch very slowly spreads. Its appearance is liable to give rise to a sense of grating in the movements of the joint when the surface is exceptionally dry, but, as Mr Redfern long ago pointed out, this change may make no sign of its existence. Thus, to give one example out of many, a bricklayer's labourer, of thirty-five years of age, suffered a severe accident, crushing his leg, so that amputation of the thigh was required immediately after his admission to Guy's; we found the cartilage of the knee-joint much worn by this erosive process. On questioning the man he declared that he had never experienced the slightest sign of disorder in the knee; his occupation consisted usually of carrying hods of bricks up a ladder, and he had no idea that his lost knee had any fault about it; it had, indeed, always served him so well that the poor fellow would hear nothing against it. Microscopic examination of the cartilage under these circumstances shows the inactivity of the change. It is true that some multiplication of the nuclei of the cartilage cells may generally be seen, but we think too much has been made of this part of the proceeding. The most striking change is the fibrillation of the interstitial substance and separation of its normally homogeneous mass into bundles of the fibres so arising, while the cells do not show at all in these fibres, but appear to have very early burst and discharged their contents or to have been wasted away. This change is usually described as proper to rheumatic arthritis, but we think it is a general process, the result of repeated irritations imperfectly recovered through the insufficient blood supply.

Sometimes the cartilage when so affected is said to be ulcerated, and we have often seen inexperienced persons misled by it when examining joints in rheumatic fever. Finding acute synovitis, with liquid exudation and lymph in the joints, associated with this degenerative change, they set all down alike to the rheumatic fever, and describe this as having caused "ulceration of the cartilages;" but this is certainly an error. The change found in such cases is exactly the usual erosion on the worn parts, and corresponds always in its extent with the elderly age of the subject; also, when other joints not inflamed in the same case are examined, they present the same erosion.

When considered on principles of general pathology the change in question shows successive stages, that is, in the irritative multiplication of the cartilage cells and the subsequent disintegration of the tissue. These same proceedings are found in ulceration of other textures, but the behaviour of the vessels in vascular parts is, of course, omitted in cartilage. Now, the active characters of an ulcer, such as we see on the skin or mucous membranes, are almost altogether dependent on the vessels, which swell and become choked with corpuscles, so as to induce death of the parenchyma dependent on them, while white and, perhaps, red corpuscles exude from their walls. The course of the ulcer, especially its healing, depends on the production of new granulation matter, containing new loops of vessel which the vascular tissue supplies. A tissue wanting in vessels and in the acts done by vessels wants also the characters by which all that we mean by an ulcer is substantiated, and it is more convenient to call the change in the non-vascular tissue by another name. The name "degenerative erosion" will serve well. For, although it has an active stage, and its cause appears to be direct irritation in the course of the use of the joint, nevertheless its leading characters are in the failure of repair, and change to a lower or less special type of mere fibrous tissue, which constitutes degeneration.

Although the cartilage is dependent on surrounding structures for its vascular supply, yet it does not follow that any obvious change in these accompany its degeneration; for cartilage has its own proper self-nourishing power, and it is to the irritation and exhaustion of this that we must look for the explanation of its failure to maintain its integrity. Cartilage, like other textures, varies in its self-maintaining faculty in different people, so that in some it is sooner overpowered, just as in some people the head sooner becomes bald and the mouth toothless save for the dentist's assistance earlier than in others. Such failures, due to original weakness of constitution, even though that weakness leads to some irritative phenomena, are really degenerations.

We have already said that in many cases of caries of the spine the disease begins in the *intervertebral substance.* Softening may be constantly observed in persons who have been long bedridden. By thrusting the point of a scalpel in between the vertebræ the elasticity is found to be lost, and, if microscopically examined, the cells are found to have undergone a fatty degeneration. The fibrous tissue is seen dotted with granules, and the cells between filled with fat-globules.

Eburnation of Bone; Ivory or Porcellanous Change.—Like the last-mentioned change, this is sometimes described as proper to chronic rheumatic arthritis; but we often meet with it in joints which are otherwise normal, and hence regard it as of the same nature as the degenerative erosion of cartilage to which it usually succeeds. The bone exposed, when the cartilage is removed, is hardened and polished on its surface by the friction in the joint. Such surfaces have been spoken of as eburnated cartilage, but there is no trace of cartilage to be seen in them. They are made of indurated bone, in which microscopic examination reveals no structure or only traces of it, so proving that mere mechanical pressure is the cause of the hardness, and that the layer should not be described as a development of a new bony surface. It is true that this change is most largely developed in chronic rheumatic arthritis, but it is not limited to this; it is sometimes found after carious disease of the joints, and often when there is no other sign of imperfection in the joints.

Injury.—Traumatic arthritis is constantly treated by the surgeon; the joint heals favorably under judicious management, though often the result is less fortunate. We have already spoken of the effects of acute arthritis, and alluded to the peculiar behaviour of the cartilage. In wounds of the joints the synovial membrane closes by a tissue resembling its own structure; but cartilage is not replaced by new tissue of the same kind, but by one simply fibrous. You will see in these two or three specimens of fissures in the joints that fibrous tissue unites the broken cartilage. Mr Poland drew attention to the curious fact that in the knee-joint the bony part of the patella may suffer compound fracture down to the cartilage and yet the cartilage remain unbroken. While speaking of injuries to cartilage we may mention that in fractures of *costal cartilage* the union is never by new cartilage but by fibrous tissue, which, as the patient advances in life, may ossify. Such injuries are rare, but the few instances in museums exemplify union by bone. The same occurs in the laryngeal cartilage—a fibrous union with occasional subsequent ossification.

DISEASES OF BURSÆ, TENDONS, AND MUSCLE

BURSÆ

Bursæ are serous sacs which form over bony prominences upon which great pressure is exerted; thus, a common situation for them is over the tuberosities of the ischia, or over the vertebra prominens or the acromion in porters, or more especially over the patellæ in those who work in a kneeling posture. Other bursæ are more deeply seated between great tendons and the bones they move upon. Any of these bursæ may inflame through excess of the pressure which caused them, or else, perhaps, through some rheumatoid tendency, and thus a swelling may form in one of the situations mentioned or beneath the deltoid muscle; or about the hip-joint, over the great trochanter, or under the psoas, or under the gluteus maximus; or about the knee-joint, under the extensor tendons, or around the semi-membranosus muscle, or under the ligamentum patella. These sacs undergo similar morbid changes to those already spoken of as occurring in the joints, but not in so regular a manner, seeing that their lining membrane is not so perfect. The sac may become filled with a large quantity of serum, or by a chronic inflammatory process a lymph may be thrown out which organizes until the whole sac is converted into a solid tumour; although the centre is generally then filled with a soft lymph or serous fluid. Sometimes, by a similar process, the lymph may form bands, which may be seen passing across the bursa, and become at last like hard tendinous cords; or its inside may be covered with shaggy tufts or pedunculated bodies, as seen in the joints, or filled with melon-seed bodies.

SHEATHS OF TENDONS

Inflammation.—The sheaths of tendons are liable to the same affections as the joints; they may inflame and produce the various ordinary inflammatory products. We have already remarked that the sheaths of adjacent tendons may share the acute inflammation of the wrist or other joints when severe rheumatic, or more especially when pyæmic, inflammation has occurred in those joints. Under these circumstances we have seen several of the sheaths filled with lymph or pus. An acute inflammation of the sheath with its enclosed tendon sometimes follows severe strains or punctured wounds, &c. The sheath then becomes filled with lymph, while the parts around swell greatly and the tendon may slough if the case be severe. The "thecal abscess" that thus arises may prove a formidable disease, bringing with it the risk of purulent absorption and pyæmia.

Ganglion.—By a slower process a large quantity of synovia or serum may collect within the sheath of a tendon, and thus a pouch of fluid may form a "ganglion," as you see in this preparation. Such circumscribed cysts may be distinguished as *simple or localised ganglion;* they are most common about the back of the wrist or on the tarsus, and being limited in size are not generally of serious consequence. But sometimes the great synovial sheaths about the flexor tendons of the hand or foot undergo a chronic inflammation and so form *compound or diffused ganglion;* the character of the inflammation then resembles very much that seen on the synovial surface in chronic rheumatic arthritis; thus, the interior of the sheath becomes shaggy with pedunculated growth and fringes, like those we have described as occurring in the joints, and if these become detached they fill the ganglion with hundreds of small bodies like melon seeds.

TENDONS

Inflammation.—Besides the acute inflammatory affection which they share with their sheaths the great tendons are subject to a chronic inflammation, which is well known clinically under the name of rheumatism. But this chronic inflammatory state does not, so far as we are aware, produce any anatomical change beyond slight thickening. The crackling sensation which is met with in such cases appears to be due to the gelatinous effusion in the areolar meshes which you commonly find about such inflamed tendons. In *injury* the toughness of tendons is exhibited by their often resisting the most violent force, so that they separate at the attachment to the muscle rather than break across; thus, the quadriceps femoris will tear from its tendon more or less completely, and you will see in these specimens how, when the fingers were torn off, their tendons were drawn quite out of the arm. Chemical and vital changes they equally resist, so that in burns or sloughing wounds, or in the removal of a part by gangrene, you still see the tendons in the dead portion comparatively little altered. Those most apt to *rupture* from violence are the long tendons of the biceps, the ligamentum patellæ, tendo Achillis, &c.

Repair.—After tenotomy the cut ends of the tendon are drawn apart to some distance depending on the length of the contractile fibres of its muscle; this distance may be as much as two inches in the tendo Achillis, for the gastrocnemius naturally traverses a large space, crossing, as it does, two joints and having a large action on both. The interval is soon filled up by a soft material, which is formed by the surrounding parts, especially the sheath of the tendon which becomes congested at the same time. The tendon itself is almost evascular, and takes little share in its own repair, its part consisting in a swelling of the cut ends and a production of lines of new material between its fibres in continuity with that in the breach, by which means the connection between the ends is made firm. In this specimen of tendo Achillis you will see a soft material joining together the divided ends twelve days after the operation, the microscope showed well-formed nucleated fibres lying together in the bundles. Mr W. Adams has seen capillary vessels in the new matter about the eighteenth day, and in three or four weeks the required length of new tendon is formed, though yet not very tough. For a long time, if not permanently, the new part differs from the old in being less lustrous, and also in having a union with parts around which prevents it from retracting so much a second time if it is again divided, but it is composed of strong fibrous tissue like the original tendon, of which it well serves all the proper purposes.

Ossification.—This is occasionally observed in tendons; we have met with an example of sesamoid ossification of both heads of the gastrocnemius, such as that mentioned by Lobstein and others, and quoted by Virchow. The last-named learned author gives a full list of the seats in which these ossifications have been seen, as follows:—In the triceps and biceps brachii, digastricus (this also we have seen), rectus abdominis, adductor magnus, and diaphragm. But the most important examples are those called the *rider's bone* and the *drill bone;* the former appears in the form of a rough growth of bone, continuous with, or attached by ligament to, the outer face of the pubis or ischium, among the fibres of the adductors. Mr Bryant gives a case in which he witnessed the formation of such a mass in a habitual rider. Drill bones were especially observed by Hasse in Prussian recruits, eighteen out of six hundred of whom had a growth of bone of variable size in the tendon of the deltoid from frequent injury of this part by rude contact of the rifle during drill.

Club-foot.—We shall only allude cursorily to the subject of club-foot, for it is fully described in the surgical lectures. Essentially it is a nervous disease, and the morbid anatomy of the nervous change which induces it is unknown. It is, however, occasionally associated with spina bifida; it is frequently congenital; it also frequently supervenes during early or even adult life, through paralytic or spasmodic affections, sometimes following severe fevers, sometimes coming on through partial paralysis. Thus, we recently had under our care two children of one family, each of whom became a subject of *talipes varus* between the eleventh and thirteenth year, through spinal weakness; a third child at nine was beginning to show the same affection. Here, although the weakness and deformity supervened late, there was reason to believe the cause was congenital, for the parents of these children were cousins. Such consanguinity of parents is no doubt a frequent cause of club-foot. The malformation is also hereditary in some families, the males being more frequently subjects of it in those families.

Club-foot arises from a loss of that balance between the several sets of muscles around the joint which preserves the joint in the position suited to its function. The balance may be lost through either spasmodic over action, or paralytic under action of either set of muscles. Which of these it is due to, in general, is a question that has been a great deal discussed, because it affects the indications for treatment very obviously. But we must not rest too certain that in all cases there is such a loss of balance of muscles. For when all the muscles of a joint equally put forth their power on it, the result is not always that the joint is in a neutral posture, but it may be bent in a constant direction. Thus, in *rigor mortis* the position of the foot is one that, if permanent, would constitute a slight amount of talipes varus and equinus; the heel being drawn up, and the sole of the foot turned inwards. If such a condition of equal action of all the muscles supervened during life, the effect would be to induce talipes equino-varus, although no set of muscles was either paralysed or in greater spasm than the rest; the position being the result of the natural preponderance of the power of one set of muscles over the others. It is to be noted that the most frequent kind of congenital club-foot is varus; the most common kind of acquired talipes is equinus; while talipes valgus is a rare affection.

New growths.—Tumours sometimes spring from tendons, but such an origin of them is not common. We have several examples of *carcinomatous* tumour, one arising from the sheath of the tendon in a finger; another from the tendons in the palm of the hand; another from the biceps femoris, &c. We have also some specimens of *sarcoma* arising from aponeuroses. Here is an *enchondroma* which appears to grow from the extensor tendons. In most cases, however, tumours on the tendons are found to be of the nature of *indurated bursæ*.

MUSCLE

Malformation.—Extra muscles occur under certain conditions, and are pointed out by anatomists; or muscles may be sometimes absent, as in a case which occurred here several years ago, where the pectoral and adjacent muscles were wanting.

Injury.—When a muscle is injured or destroyed it is repaired with a fibrous structure, and never by the reproduction of muscular tissue; if such a muscle be put to much use the fibrous structure assumes the consistence of ligament. *Spontaneous rupture* may occur from violent spasm, as in tetanus, of which Mr Curling has given many examples. We have seen the rectus abdominis thus torn; but rupture in tetanus is more common, we think, in the muscles of the back, where you will often find laceration and effusion of blood. Rupture of muscle occurs also during violent effort in delirious persons, or from accidental injury suffered by them in unguarded movements made during delirium. This is, no doubt, the cause of some of the ruptures of muscle which we find in the bodies of persons dead of fever; but the changes we shall presently have to describe as occurring in the muscles in typhoid may have a share in causing the ruptures. We have met with suppuration of a ruptured rectus in typhoid, an abscess being formed between the ends, where the muscle was completely torn across and showed abrupt ragged edges. In the cases we have seen, the rupture has usually been through the fleshy part of the muscle; but we have twice known the extensors of the thigh partly torn from their tendon. The other muscles which have been known to rupture are the biceps femoris and gastrocnemius.

Inflammation.—This, when attended with the usual results, as suppuration, gangrene, &c., is by far most frequently pyæmic, or due to thecal abscess, or other neighbouring active disease; but the muscles will suppurate spontaneously. Thus, we have sometimes met with psoas abscess where only the muscle was affected, and we have had two or three examples of great abscess in the deep gluteal region extending into the pelvis through the ischiatic notch when the bone was not affected, and the muscles were the parts principally destroyed. Suppuration arises and proceeds in the cellular tissue among the fibres, and the pus-cells may be seen in lines between the muscular fibres before these are themselves involved. The effects of chronic inflammation we shall presently speak of under degeneration.

Hypertrophy.—*Hypertrophic paralysis.*—Hypertrophy of voluntary muscles does not become a morbid condition. When well pronounced, as in the blacksmith's arm, it does but show what size the full nourishment of the muscle will bring it up to. A very interesting condition of muscles has been pointed out by Duchenne, of Boulogne, under the name of hypertrophic paralysis. It is found generally in young persons. In the few cases we have seen there was general, incomplete, muscular paralysis, affecting, indeed, all the muscles, and associated with the usual wasting of most of them; but some of the muscles, especially the gastrocnemius, glutei, and erector spinæ muscles, instead of wasting, had remained large, or had even increased in size beyond their normal volume, while they were as much paralysed as the others. We have never examined any case of this disease post-mortem; but in a little piece of such muscle removed during life the apparent hypertrophy was found as usual to be spurious, and due to the presence of fat-cells between the muscular fibres which themselves were wasted away.

Degeneration and Atrophy.—These are usually chronic processes, and are generally of the fatty or fibrous kind, though pigmentary degeneration sometimes occurs in the heart, and a waxy degeneration is spoken of in progressive muscular atrophy, which, whatever its nature, must not be confounded with the waxy lardaceous change in the liver, &c.

Fatty degeneration.—This degeneration shows itself as a deposit of fat-grains within the muscular fibres. It is usually a chronic process, but we have seen an acute fatty degeneration in a case of poisoning by phosphorus, the muscular fibres not only of the heart but also of the voluntary muscles being very much charged with fat-grains, so that the transverse striæ in many could not be seen, and this although life was not prolonged more than eight days after the poisoning. Others have made the same observation. This acute fatty degeneration, or *steatosis* as it has been called, implicates also the liver and kidneys; in arsenical poisoning we have verified its occasional occurrence; it has also been found in poisoning by antimony as well as in acute atrophy of the liver. In all of these conditions the disorder is of short duration. Such rapid development of a change once thought to be always chronic is a suggestive fact.

The production of fat-grains in the fibres of voluntary muscles is not so frequent as in the muscle of the heart, where it is so often noticed. The proportion of fat present when this change is microscopically well marked is very small, not more than 5·7 per cent.; and, indeed, this granular fatty change of the fibres never much alters the red colour of a muscle, at least, of voluntary muscle.

But you often see an evident fatty change in the voluntary muscles of old people, particularly of those long bedridden, or in the bodies of old paupers in the dissecting-room who had long fed on workhouse diet. In such bodies you may see the whole muscle, say a pectoral, apparently almost changed to fat; in another case streaked with this fatty change; or, yet more frequently, you see this fatty change in the long disused muscles around an old scrofulous hip- or knee-joint. The microscope will show you the nature of this change, and you will observe that it is a totally different condition to the granular degeneration we have been speaking of. What looks like fat is really histological fat, the proper cells of which are found insinuating themselves between the muscle fibres, at first chiefly in the course of the vessels, afterwards in all parts, so that they waste away, a wasting which the disease that induced the formation of the fat much encouraged, and, perhaps, indeed chiefly caused. But disease is not the sole cause of this fatness of muscle, for it is habitual in over-fattened animals. The wasting of the muscular fibres under these circumstances is generally simple and not accompanied by any very marked granular change.

Fibrous or fibroid degeneration.—This is also often found in the neighbourhood of chronic inflammation, hence it is termed myositis by some authors. Portions of muscle around carious joints, which in other parts show the fatty state above described, are often seen as a hard white structure in which the unaided eye perceives the remains of muscular fibres visible by their linear arrangement. This fibrous change is the natural result of chronic inflammation in muscle, and is found after repeated rheumatic attacks and after syphilitic inflammation. The change is best known in the heart. The muscle is converted into a tough semitranslucent white substance, which, when the change is complete, is found microscopically to be well-formed fibrous tissue. The earlier stages are not well known. They are best studied about old knee-joints, and near periosteal inflammation in the deep muscles of the thigh and leg. The new material follows the vessels, so that a corpuscular new formation surrounds them. We never could decide whether the corpuscles arose through exudation or through multiplication. But as to the permanent new fibrous tissue we believe it to take its form from the pre-existing fibrous texture, as, indeed, all scar-tissue does, while the new corpuscles of the inflammatory act are probably derived from the vessels, and do not take part in the healing process that forms the fibrous scar.

Besides such fibrous degenerations, which are probably inflammatory in their origin, there are others that are certainly passive. Thus, the over-stretched columnæ carneæ of a dilated left ventricle becomes changed to fibre.

In progressive muscular atrophy or Cruveilhier's paralysis the muscles waste away because of a degeneration of the cells of the anterior grey horns of the spinal cord. The tissue of the muscle in the several cases we have examined has shown little more than a simple wasting. The electrical excitability, you will remember, persists in this disease as long as any fibres of the muscle are preserved. Dr Lockhart Clarke describes a fatty, fibrous, and waxy degeneration of the muscle in this disease; others also describe a fatty change. We would not deny that some fibres may be found so affected, but it is remarkable how simple the atrophic process commonly is, the fibres growing smaller and smaller, still preserving their striæ until the then remaining parts break into yellowish grains.

In muscular wasting through other paralytic nervous diseases the same is true, as far as we have been able to see; the wasting is a simple dwindling with little alteration of structure. We have not been able to satisfy ourselves of the irritative proliferation of the sarcolemma described by Erb as occurring in muscles whose nerves are artificially destroyed, and by Mannkopf in muscles wasted by acute myelitis. These authors are quoted by Charcot to prove that such signs of inflammatory irritation in the muscles accompany the loss of electrical contractility in irritative diseases of the spinal cord and nerves. We would, however, mention a curious incident, bearing upon this question, that recently occurred in the case of a man who had intra-cranial cancer destroying the spinal accessory and pneumogastric nerves. This man had not only complete paralysis, but great wasting of the sterno-mastoid and trapezius muscles, notwithstanding that the spinal supply to these muscles was intact. The usual explanation of wasting of the muscles by supposing it due to a loss of nervous influence is obviously inapplicable here, for the spinal supply of the muscles was quite perfect. Hence the case gives a certain support to the belief that a morbid irritation of a nerve-trunk, in this case the spinal-accessory, directly and actively damages the nutrition of the muscles supplied by it, and so leads to their wasting. Still we could not be sure that the connective tissue in the muscle had increased. This is a very doubtful observation at best, for a relative increase, of course, occurs when the proper muscular tissue wastes.

Waxy degeneration (Myositis Typhosa).—We have already mentioned the occurrence of *abscesses* about ruptured muscles in fever. Abscesses have been long known to occur in fever, and were looked upon as part of a pyæmic process until Zenker drew attention to certain changes which parts of muscles sometimes undergo in typhoid. This condition has received much attention in Germany, but little has been discovered in England concerning it; we have not been fortunate in meeting with examples of it, unless some incipient abscesses, one in rectus femoris, one in rectus abdominis, which we should have ascribed to injury, were really of this nature. It is not peculiar to typhoid, but appears also in cholera, acute tuberculosis, smallpox, and other diseases.

The change is said to affect chiefly the adductors of the thigh, the abdominal and pectoral muscles, and the diaphragm, as a circumscribed patch from one to three inches in diameter, in which the fibres are seen swollen and waxy, or so softened into a reddish pulp that the whole looks like a muscular abscess. The appearances presented microscopically are those of acute inflammation; the sarcous substance swells, and loses its striation, becoming translucent, as if from fusion of its sarcous elements; inflammatory corpuscles meanwhile appear between and within the fibres. Rindfleisch says these cells show a peculiarity in their large size, which allies them to the proper cells of "typhoid deposit" in the intestine. After a time the old fibres waste away, and new ones appear inside the sarcolemma tubes in the form of long polynucleated spindles, such as are found in the original development of muscle.

It would be interesting to inquire whether the phlegmasia dolens, which occasionally follows typhoid, has any connection with this myositis typhosa in the adductors of the thigh.

Tubercle.—The occurrence of tubercle in muscle is very rare, and we have no specimen to show you.

Syphilitic disease.—This, on the other hand, is very important, and not very unfrequent in the muscles. It might easily be mistaken for tubercle when in the caseous form, with softening in the centre. Syphilitic inflammation of muscle is found showing characters according to the degree of intensity and the stage of inflammation, as well as the extent of structure implicated. It is only rarely now, since Bouchier drew attention to their nature, that we meet with examples of syphilitic formations removed by excision from the muscles of the limbs; such occurrences were formerly by no means rare. When so removed we have seen them in the form of *caseous* masses; one was as large as a hen's egg, the interior softened down. The microscopic structure of caseous syphilitic myositis shows the usual characters of syphilitic disease, *i. e.* a highly corpusculated lymph, whose elements resemble those of ordinary granulation, and tend rapidly to decay. Sometimes instead of a cheesy mass we find a syphilitic *fibrous* tumour, not quite like other fibrous tumours, which are circumscribed, and displace the muscle in their growth; but rather formed between the muscular fibres out of a lymph developed interstitially, so that they may often be seen to contain the relics of muscular fibre within them. These syphilitic tumours are found more often in the tongue, and in the muscles of the legs and forearm. We have seen them in the sterno-mastoid muscle, and have examined a specimen removed from the deep muscles of the thigh. Besides these *circumscribed* masses, which would, at first sight, be called tumours, syphilitic myositis appears in a *diffused* form both in the heart and in the muscles of the limbs. Indeed, it is most important to be well acquainted with the effects of syphilis on the muscles of the limbs. We have met with cases which had been called progressive muscular atrophy, and, indeed, closely resembled this terrible disease, yet which rapidly got well under iodide of potassium in spite of the grave prognosis which had been given. Such cases of general wasting however, we think, are due to syphilitic nervous rather than muscular disease.

Morbid Growths. *Cancer.*—The muscles are frequently invaded by malignant tumours arising in the breast or the bones. Also in the lips and the tongue the spread of epithelial cancer from the mucous membrane or skin into the muscle is frequently enough met; but *primary* tumour of muscle is rare. Mr Teevan has carefully collected many cases of primary cancer of muscle, of which nearly all were called medullary cancer. The two exceptions called scirrhus are doubtful. Of the few cases we have seen one in the right rectus abdominis was medullary sarcoma. The term medullary cancer, as used in the description quoted by Mr Teevan, would not be distinctive between different kinds of rapidly growing malignant tumours, which are all apt to be called medullary cancer whether they be sarcoma, lymphoma or carcinoma. From a practical point of view this is of no importance whatever, and, indeed, the lines of distinction between the forms of growth we have named reach their vanishing point in the vague histology of such tumours as these soft, swiftly-formed, malignant cancers of muscle, whose structure does not offer particulars enough for useful distinction.

It is remarkable that all the cases Mr Teevan collects occur in the larger and more superficial muscles. This, however, may only be because tumours in deep and small muscles would not have been sufficiently recognisable and removable at the stage when they are limited to the seat of their origin.

When cancer does invade muscle or arise in muscle its spread in it is generally free. In the cases we have examined from the neighbourhood of cancers in breast the growth has been found extending between the muscular fibres. What ultimately becomes of the fibres themselves it is very difficult to see. We do not think it can be confidently said that the proper sarcous substance has no part in the formation of cancer. It is very certain that if the muscles be examined around a spreading cancerous tumour, nucleated cells may be found in parts which are apparently healthy to the naked eye, and these are always appreciable with facility, but more especially in the tongue can they be discerned, because the cells and nuclei are of large size, and often contained in compound cells, the cancer being of that kind which is called epithelial. In such a case very often, if the muscle be examined for some distance around the disease, these large cells may be met with in the course of the fibres, a microscopic sketch of which you will here see. For we have seen the muscular fibres, though at first resisting the growth and merely compressed by it, at length, that is, at a greater depth, appear to divide up into spindle-like bodies, each with a nucleus behaving, indeed, like the hepatic tissue behaves in presenting intermediate stages of transformation from the normal elements to cancer-cells. In epithelial carcinoma of the tongue we have convinced ourselves of the growth of cancer-cells within the muscular fibres. This is often well seen. It has been insisted on by Weber, the accuracy of whose drawings we can fully verify. It has been urged by some that the pressure of the cells indenting the fibres would produce the appearance of their entry into it; but the appearance of cancer-cells within the fibres is so frequent that this explanation will not hold good.

It is important to notice the free spread of cancer in muscular tissue. We recently examined the line of section of a cancer removed by galvanic cautery from the tongue, and found cancer-cells present close up to the surface of excision, so that no doubt could be entertained that cancer-cells had been left, though the part had to the naked eye all the appearance of being perfectly healthy. The practical importance of this is evident. It is necessary to remove a muscle largely around any cancerous spot within it. Mr Teevan urges the necessity of always taking away the whole of any muscle that has been invaded by cancer, and, no doubt, this is a good rule so far as it is practicable.

Fibrous tumour, &c.—The other primary tumours in Mr Teevan's list were generally described as fibrous; we have examined such tumours; one from the semi-membranósus was a fibro-sarcoma, the fibrous element predominating. There were no other tumours in the body. No case of *lipoma* is recorded, and other forms of tumour are rare; but Mr Teevan collects five examples of erectile (cavernous) tumour in muscle. *Cysts* are not often met with in muscles. In one we lately saw the origin was probably a softening of one of the fibroid masses just spoken of.

Parasites—*Trichina spiralis.*—The entozoon peculiar to the muscles is *Trichina spiralis.* This very old specimen, preserved in the museum, contains many. But the nature of the affection was unknown at the time when it was put into our collection. It appears that Mr Hilton first suspected the little grains might be parasites, and sent them accordingly to Professor Owen for determination, by whom they were recognised as worms and named with the name by which they still are called. Trichina spiralis is very infrequent in this country. In our own experience we have not met with it oftener than once in five hundred bodies examined by us, if so often as this. The affection is known by the voluntary muscles appearing to be marked throughout with countless white specks. When more closely examined the whole tissue is seen to be pervaded with little white oval bodies; they are hard, and always lie with their long axis in the direction of the fibres; they are placed at regular intervals not crowded into masses. If picked out and placed under the microscope they resemble miniature eggs; the shells or cysts are composed of earthy matter and are very hard, requiring some force to crush them. When one is broken carefully a round worm emerges; it is pointed at the ends, especially the caudal end, and it is the encysted larva of a small nematoid which would reach its sexual perfection in intestines of flesh-eating animals. These creatures are distributed in all the muscles with striated fibre, except the heart. Virchow is said to have seen them in the heart, but the usual rule is shown in this preparation, where the heart is free, and yet the pharynx has its involuntary striated muscle full of them, like the biceps, which has been chosen here to represent the general muscular system, though muscles of the trunk would have served better, since they contain more trichinæ. When thus encysted, as they have hitherto been found in this country, there has been no history of any ill effects from them. It appears from the observations of continental pathologists, especially Virchow, Leuckart, and Davaine, that it is only when the worms are moving to take up their positions in the muscles that the serious and, perhaps, fatal mischief they cause is observed. The following is a short abstract of their history as given by the last-named author. The sexually mature worms inhabit the intestines of many kinds of animals; they very quickly reach their maturity on entering the intestines. They are viviparous, and their young, soon after birth, begin wandering through the intestinal wall, penetrating on their way the peritoneum, and working on until they reach the voluntary muscles in whose interstitial connective they must lie. Their progress excites a traumatic irritation, which locally produces a granular change in the fibres with serous effusions, pains, and paralysis; besides this they give rise to exhausting fever and, perhaps, peritonitis, from either of which the sufferer may die.

The symptoms are in proportion to the number of the creatures. In their position in the muscular flesh they wait the time when their host may be eaten, and it appears that in that larval state they are very tenacious of life, though they are very perishable when adult.

Cysticercus (*telæ*) *cellulosæ*.—This entozoon is the cystic worm whose presence constitutes "measles" in pork. Though not frequent, it is far from very rare in man. Its more usual place is the cellular tissue of the muscles; but it is found in other parts. Thus, Griesinger collected more than fifty examples of cysticercus in the brain. It is generally of small size, not larger than a horsebean or hazel nut. It consists of the cystic caudal extremity of the animal with the head and neck coiled spirally and embedded in a spot on its surface.

The *Echinococcus* or true hydatid may be found in the muscle as in all other parts of the body.

DISEASES OF THE HEART

PERICARDIUM

Malformation.—The pericardial sac has occasionally been found altogether absent. Some of the old writers had already observed this fact, but discredit was thrown upon their statements on account of the very frequent obliteration of the pericardial cavity by adhesion, and which was thought a sufficient explanation of the apparent absence. This supposition might have been true in many instances; but, at the same time, such a want of development does sometimes occur; for, a few years ago, we had an opportunity of seeing a specimen of the kind, brought to the Pathological Society by Dr Baly, where the heart was uncovered, and lay in the cavity of the pleura against the left lung.

Hydropericardium.—The pericardial sac is liable to simple serous effusion like other serous cavities. The effusion is said once to have amounted to eight pints, but it is usually under half a pint, and then forms part of a general dropsy. A certain quantity of fluid is found in the pericardium in all cases when the body has lain some time after death. But the amount is not large, not more than an ounce or two. If any more than this be found its cause should be searched for in some obstruction of the vessels; such an amount as six ounces, often quoted from Corvisart as possibly normal, would certainly be traceable to disease. Mr Hilton proved it to be due, in some instances, to absence of the *valvula Thebesii*, which should guard the orifice of the coronary sinus. Active catarrhal flux into the pericardium is said to occur sometimes. We have never met with it as an independent disorder.

Inflammation—*acute.*—In ordinary acute pericarditis the earliest stage is seen as a minute injection of its vessels, causing a blush of redness, which close observation resolves into a beautiful red network. This injection is almost a certain proof of pericarditis, but when you see it you should look at the base of the heart, about the great vessels, where you will always find some shreds of inflammatory lymph. This earliest stage is soon over; in a day, or two at most, a fibrinous layer is formed, which, in the course of a few more days, forms a complete covering to the heart, and which, if there is no fluid exudation, may connect the surfaces of the heart and its sac rather firmly. This forms the second stage or degree of pericardial inflammation. We more frequently see some serous exudation mixed with the solid lymph, and thus the latter is found in shreds, or presenting a honeycomb appearance, very much as if a layer of butter had been placed in the sac and the surfaces suddenly separated; but when the quantity of fluid is small you will notice that the parts of the heart which are kept in contact with the pericardium show a surface like that of a file, with rugosities across the direction of the friction which has caused them; such roughness of the lymph gives rise to the rough, rasp-like, friction sound which accompanies pericarditis of this third degree of severity.

Very early pericarditis is generally met with as a complication of morbus Brightii, but rheumatic cases generally survive until the disease is more advanced, and then a thick layer of lymph will be found covering the heart; and if there be much fluid effused with it, an occurrence not generally met with until a much longer period has elapsed, and more proper to fatal rheumatism, then the more solid part puts on a very remarkable appearance. From the two serous surfaces not touching each other, shreds of lymph float in the fluid, and the surface is, consequently, covered with long shaggy processes. This is the condition we generally have in our minds when we speak of acute pericardial effusion as it occurs in rheumatism.

Occasionally the inflammation, through either long duration or intensity, or else through the low state of the patient's powers, is purulent, and thus, in the same way as a pleurisy ends in an empyema, so may a purulent effusion result from a pericarditis; it is not very common, and no doubt is generally fatal. It usually owns a cause different from the cause of either of the fibrinous forms we have described; we have seen it once when an aneurism had approached and compressed the pericardium. The bursting of hepatic or other abscesses into the pericardium may cause it. It is sometimes idiopathic. In one case, of which this is a drawing, you see the pericardial sac immensely distended, and within it were thirty-six ounces of pure pus: the man had been ill two weeks.

In some severe cases of acute pericarditis, the heart's muscle beneath the pericardium is found to undergo a fatty change to a depth of one sixteenth to one eighth of an inch, the amount of fat in the muscle fibrils being so large that the layer becomes conspicuous to the naked eye as a yellow line under the pericardium.

No doubt in very slight cases the new lymph will disintegrate and complete recovery by resolution will be the fortunate result. Severer cases undergo simple adhesion of the opposed surfaces, with little or no bad consequences. In the graver cases the adhesion is usually complicated by the effects of inflammation on the heart, and only helps to sum up the consequent *morbus cordis.*

Chronic.—As we have already had occasion to mention, the so-called chronic inflammations are often altogether different in kind from the acute, being slow in their origin as well as progress, the process of production of new material being more allied to that of a growth than of an exudation which has subsequently organized. In the pericardium, however, most of such chronic changes have an acute origin; for example, after a pericarditis and effusion of lymph which organizes, a continued succession of exudatory layers takes place, and if the case be fatal within a few weeks or months, the various stages of the process are seen. Thus there may be a firm layer of fibrin adherent to the heart, and over this a softer and more recent exudation. The heart's substance will be found more or less implicated; we have mentioned above the effect of acute pericarditis in causing acute fatty degeneration of the muscle beneath. In chronic cases you may find a growth of fat beneath the membrane, with or without an extension of fibrous tissue into the superficial layers of muscle.

Occasionally, however, though much more rarely than in the pleura or peritoneum, chronic changes may be met with which seem to have had no acute commencement. Thus the visceral pericardium is much thickened, but unadherent, involving the heart in a fibrous layer, uneven on the surface, and encroaching often on the muscular texture of the organ itself. The fibrous patches are found spread irregularly over the surface. From the absence of adhesion it is difficult to believe that true inflammation occurred in these cases; the change is exceedingly slow. You will often find the fibrous tissue dipping into the underlying muscle of the heart.

Adherent Pericardium—*simple.*—As a result of pericarditis, the serous surfaces may become wholly or partially adherent, or the membrane merely thickened in particular spots. Thus, several years after an attack of pericarditis which has been cured, an adhesion may be found attaching the front or other part of the heart to the sac; not uncommonly the apex is thus adherent, and sometimes a long loose cord is seen attached to it, as of a band which had broken. More frequently than this, the heart is found universally adherent to its serous covering by means of a most delicate areolar substance, and then no evil appears to result from it; thus we found it three times in one week on one occasion, and on another four times in one week, always innocent of any share in the fatal illness. We cannot, therefore, but think that its occurrence is quite unconnected with symptoms, or any impaired action of the organ. You might easily suppose that, if nature has made the heart to move freely in a serous cavity, the obliteration of this would of necessity impair its motions; but you might also think the same of the lungs; these organs, in like manner, are intended to be free in the chest, and yet adhesions appear to do no harm, and are found, more or less, in nearly all cases we examine. There may be, indeed, some amount of impairment of action in either case, but this is not appreciable, nor are any ill effects recognised; indeed, when we reflect that no vacuum arises in the pericardium during the action of the heart, so that the sac must follow the heart's movements, the delicate adhesions would only seem to help that result. When such adhesions were first recognised (at the time of the discovery of auscultation), it was thought that an enlargement of the heart resulted therefrom, and no doubt the two conditions may often be found combined; but in many of these cases it will be seen that valvular disease is also present; in others the enlargement might be considered to be due to other causes, since a hypertrophy often takes place without any pericardial affection. Our own opinion is that simple pericardial adhesion produces no appreciable untoward consequences.

Fibrous.—But there is another class of cases, the result of graver pericardial inflammations, in which the adhesion of the pericardium is by a thick vascular fibrous layer, and the muscular substance is torn by the attempt to separate the pericardium from the heart. The heart is then always found dilated and hypertrophied, and there may or may not be valvular disease coexistent, for the disease is always the result of a severe, and often of a general, carditis. To understand how such cases come about, you must remember the change under the acutely inflamed pericardium, which we have already mentioned to you. It is natural to expect that such a damage of muscle must lead to loss of power and temporary dilatation of the cavities, which dilatation, creating a greater mass of blood to be moved, furnishes that resistance which the heart is always so ready to respond to by hypertrophy, and in this way a grave pericarditis leads at last to the dilated hypertrophied condition we are speaking of.

Calcareous.—When the inflammatory lymph reaches a subpuriform state through intensity, and yet the case recovers, and life lasts long after, the pericardium undergoes a very surprising change in the form of an earthy degeneration or petrifaction. At first this change is limited to the remains of the pyoid lymph in the sac, afterwards it invades the membrane itself, and at last it will extend into the muscular substance of the heart, so that we have found it reaching, in the form of round advancing masses, through the thick wall of a hypertrophied ventricle to one tenth of an inch from the inner surface. The usual position for these "bony plates" is around the base of the heart, forming a circle, which runs just below the coronary arteries, not usually implicating these at all; it crosses the conus arteriosus at its middle; sometimes one or more round plates are found on the anterior surface, sometimes they are on the posterior surface, connecting the heart with the diaphragm.

White Patches, Milky Spots.—The question has long been under discussion, whether the white patches so often seen on the surface of the heart are inflammatory or not; some looking upon them as mere thickening of the membrane, and others as the result of inflammation. If by inflammation we mean that there has been a pericarditis, and lymph effused, which has subsequently organized, there is no proof that such has ever occurred. The obscurity of the term inflammation has probably had much to do with the difficulty in determining the point, for no doubt most pathologists are assured that such patches are of slow formation. If this be granted, it is another question how far the term chronic inflammation can be applied to such a process. Occasionally, as a result of pericarditis, some of the lymph may remain and organize, producing white thick patches on various parts of the heart, such as we have already described; but these are mostly irregular, and at base or apex, while the white patches of which we speak are always on parts of the organ, which come in contact with the sternum, the fifth and sixth ribs, or otherwise suffer most friction. Thus on the front aspect, in the middle of the right ventricle, a circumscribed white smooth patch may often be seen, also on the apex of the left ventricle in front; the same also on the posterior aspect, especially on the projecting coronary vessels, and sometimes on the right auricle. Hence we believe them to be due to *attrition*, and formed therefore in the same manner as callosities in other parts, such as corns on the toes; which are not, if of slow formation, generally called inflammatory. It is much in favour of this view that they are generally found on large strong hearts, especially the hypertrophied hearts of granular kidney cases, though here, as to the question of their inflammatory nature, we must make two notes:—First, that we have twice met with a small layer of recent lymph limited to such patches; and second, that it is just in these cases of granular kidney and large heart that we are so apt to meet that acute pericarditis we have before mentioned or associated therewith, and which it might be suggested may be due to friction on the covering patches.

Hæmorrhagic Effusion.—It occasionally happens that, on opening the pericardial sac, blood is found within, and a question immediately arises as to its source. If it be pure and clotted, it has generally flowed from a burst aneurism or ruptured heart, but if it be in small quantity, then, on closer inspection, we may find the serous surface covered with lymph, and it becomes apparent that the blood is merely an accident of pericarditis; in one or two cases lately, we have seen much importance attached to this presence of blood, and the vessel carefully looked for whence it was supposed to emanate; but it is merely an accident of the pericarditis, and depends, probably, upon some constitutional cause; for a disposition to purpura may often be observed elsewhere in the body, and hæmorrhagic pericarditis has been occasionally observed in an epidemic form suggesting an association with scurvy. The source of the blood is either from new-formed vessels, which, being yet tender, have given way; or, in the absence of proof of the existence of such, it is more likely that the vessels of the serous membrane have furnished the supply; these being highly congested, and the tissues softened. A similar sanguineous effusion is sometimes noticed in the pleura and peritoneum. Occasionally, where the lymph is solid, and the pericardial sac closely in contact with the heart, the blood is found in coagulated layers interspersed with the white laminæ of lymph.

Air in the Pericardium.—Whether air can be secreted from a closed surface during life is a question much discussed by pathologists. There can be no doubt that in most cases air found in closed cavities of the body results from decomposition; and with respect to the heart, we have never met with air in the pericardium, unless its source was clearly either putrefaction or entry from without during life through a communication established with some air-containing cavity; as once with the œsophagus or as in another case with a hepatic abscess that also communicated with the intestines, or in a third with a phthisical vomica. Of secretion of air into the pericardium we have no experience. In one case, where, owing to very remarkable sounds, it was thought air might be present, the patient recovered.

Morbid Growths.—*Fat.*—This often exists in excess under the pericardium, but its importance is connected generally with degeneration of the tissue of the heart with which it is associated, and therefore we shall refer to the subject hereafter.

Cancer.—In cases of widely spread cancerous deposits throughout the body the pericardium may also be included, and numerous white elevated patches of the deposit may be found upon it. Also, in cases of cancer about the root of the lung, malignant growths may penetrate the serous sac. They then are found in the form of nodules growing into the pericardium about the pulmonary veins. Their pressure may cause a direct disturbance of the heart's action and so add much to the whole sum of misery induced by that terrible disease.

Tubercle.—In the same way as cancer may occur on the serous sac, so may tubercles in cases of general tuberculosis. They are seldom, however, found without an inflammatory process being present at the same time, and thus the affection is analogous to a tuberculous pleurisy or peritonitis, with both of which the tuberculous pericarditis is sometimes associated when no other tubercle is found in the body, the whole forming a peculiar diathetic disease of the serous membranes to which sufficient attention has not been given. The three serous membranes often are in exactly the same state. The inflammation is essentially chronic, and so the disorder may be looked upon as one of the forms of chronic pericarditis. A good specimen is here seen; the pericardium is closely adherent, and on separating and tearing open the lymph, a quantity of tuberculous matter is seen amongst it.

SIZE AND FORM OF HEART

Malformation of Heart.—Much light has been thrown upon the subject of malformation generally, by considering what changes would be wrought upon organs by diseased conditions occurring at an early period of fœtal life, when the parts are yet plastic, ready to yield to any new influence; and how, also, other structures in connection with them would of necessity be modified to stand in relation with the altered parts. Such considerations have afforded many explanations regarding malformation of the heart. In speaking of diseases of the fœtus, remember, we do not use the term in so general sense as to imply a mere deviation from the normal or healthy standard of development, but we mean something much more definite; that is, that the fœtus becomes the subject of a disease similar to what occurs in the adult, and that in consequence various adhesions or obstructions may result which, from the parts being still plastic, and undergoing great changes during growth, produce those alterations in form which we call malformations. It is true that there are arrests of development and want of union of the two symmetrical halves of the body, and the same occurs in the heart; but still a large number of malformations appear to be due to the cause we name. That diseases do occur in the fœtus, you can easily prove by examining still-born children, and especially those of syphilitic mothers, when you will find, as we have often done, acute inflammation in the abdomen, peritoneal adhesions, pneumonia, &c.; an acute endocarditis is thus supposed to occur in the same way as in the adult. Even if the proof of this should be wanting, which we think it is not, the theory forms a good hypothesis to explain the various appearances met with in malformed hearts, and on which we can give a general interpretation of the different kinds of malformation seen on the shelves of our museum; for otherwise it would be impossible to describe each separately, seeing that we have more than thirty specimens, and scarcely two alike. We will, therefore, only show you the most common, and describe the mode in which they appear to be formed.

And first it will be best to see clearly what they are not. A general impression for a long time prevailed, that an open foramen ovale or ductus arteriosus is a common kind of malformation; this belief was founded both upon the fact that such conditions are met with, but it was too easily assumed that a malformed adult heart would very probably show this simple kind of malformation most commonly. Such opinion we still find prevailing among students, but now warn you against it. The most common kind of malformation, and that of which we have seen the greatest number of instances, is imperfection of the ventricular septum; such hearts often show one vessel coming off from the two cavities, the lungs being supplied by some irregular means; and with these irregularities an open foramen ovale or open Botalli's duct may coexist. Now, these and similar changes may be accounted for by supposing that in early fœtal life, say about the sixth or eighth week, an endocarditis had occurred (we know, from observation, that this is more liable in fœtal life to happen on the right side than the left). Thus the pulmonary valves would become affected by an inflammatory change; adhesions or other changes then would occur among these valves, causing the partial closure of the artery. After this we can infer with some certainty what the subsequent result would be. At the time in question, remember, there is no perfect septum between the ventricles, and the blood of the right freely communicates with that in the left cavity; therefore, if any impediment should occur at the outlet of either, the stream is diverted through the septum to the other, and so the closure of the septum is prevented at the proper period of its occurrence; and thus the deficiency occurs in the wall which you see in these specimens, where the pulmonary artery is contracted. Again, in consequence of both ventricles supplying one vessel, the aorta, the latter becomes placed over both of them, which are then of equal size, and the heart rounded in shape. If the obstruction be thus compensated for by an outlet into another channel, there may be no great stress on the auricle behind, and the foramen ovale may close as usual; but very often it remains patent, in order to relieve this chamber of the heart. And now as regards the supply to the lungs; in consequence of closure of the pulmonary artery the ductus arteriosus remains open, and thus the lungs are supplied from the aorta. The blood is necessarily mixed, and hence one cause of cyanosis and early death in such cases, although very often the duct is brought low down over the right ventricle, so that it gathers most of its blood from this side. You see, then, how an obstructed pulmonary artery necessitates all these further changes; a passage of blood into the left ventricle, and then a way out again into pulmonary arteries, by the fœtal ductus arteriosus remaining patent. All these different alterations in the heart would so clearly arise from an obstructed pulmonary artery that we think there can be little doubt that here lies the *fons et origo mali*. In the three or four cases of malformation of the heart in persons who have grown to adult age, the peculiarities have been of the kind we mention.

In some cases the obstruction of the pulmonary artery is not complete, and the various conditions mentioned may be less marked, as in cases where the valves unite to form a funnel-shaped membrane projecting into the vessel, while the heart is otherwise well formed. We have several specimens of this kind, in some of which the persons had lived beyond middle age. In some instances the pulmonary artery is united to the aorta, and both come off from a common ventricle; in other cases, the vessels are transposed. In some remarkable cases, although the pulmonary artery is closed or wasted away, the ductus arteriosus is also closed, as was the case from which this specimen was taken; but the lungs were supplied by immensely dilated bronchial arteries. In some other cases the aorta is obstructed, and the pulmonary artery carries the blood from both sides of the heart, the descending aorta being attached to the pulmonary. It is seen in this heart lately found in the dissecting room. We cannot mention all the other varieties met with, but you see in these preparations that the septum ventriculorum is sometimes imperfect at its upper part, without any other alteration; when this is the case, the question often is asked whether it has arisen from disease, *i. e.*, from endocarditis and softening of muscular structure, or malformation; but these suppositions are not to be taken as necessarily alternatives, for assuming that malformations arise from disease; it is possible to conceive that such disease occurred in the fœtal state, this condition being the remnant of it. We stated just now that the old notion that malformed hearts were due to want of closure of the openings natural to the fœtal state was erroneous, and you will now see how such open channels necessarily follow from previous obstruction, and constitute conservative results rather than morbid states; still, however, you occasionally meet with open ductus arteriosus without any of those obvious causes to which we have alluded, though it is possible that these causes may have existed for a time, and have afterwards disappeared, the effect alone remaining. We should mention to you, however, that a simple patency of foramen ovale is not uncommon, being met with in about one in every thirty cases. Indeed, a partial opening extends to a much later period of life than was formerly supposed; it has been found that the bodies of children show a larger proportion of examples of this condition in proportion as the children are younger, so that it is probable that the completion of the closure is a process that occurs gradually for years after birth; the opening, however, is valvular and incapable of allowing the passage of blood. We may show you this heart, where the membrane closing the opening bulges out; and this, where it is cribriform.

Here are some malformations of the heart which depend on deficient development, found only in the fœtus or children who survive their birth but a short time. Thus sometimes a heart is met with having only one auricle and ventricle. Such a bilocular heart, in spite of its low development, may show sufficiently perfect formation of the great vessels. Such cases do not grow to active life, but may continue to exist as sucking infants for a few months. Cases of trilocular heart, with two auricles and one ventricle, may reach adult life. Occasionally, too, we meet with instances where the right ventricle is divided by a septum, thus multiplying the natural number of cavities; lower degrees of this division are not very uncommon. In this case the heart protruded from the chest, owing to the absence of part of its wall, a state known as *ecstrophia cordis*.

Atrophy of the Heart.—This may arise from various causes. Some authors speak of concentric, simple, and eccentric atrophy of the heart. When the heart is wasted, if its cavity be small, this state would be called concentric atrophy; if the cavity be normal in size, simple atrophy; and if the cavity be large, eccentric atrophy. The symptoms of these states are given by some; but we have not been able to find any evidence that these conditions are of the least importance, and have never known such "atrophies" to have any share in the cause of death. It is true that the small heart of an emaciated person may be found contracted or dilated; but this depends entirely on the mode of death, so that we will not recommend you to make use of the formidable string of terms we have just alluded to. Simple wasting is found in emaciated persons as part of the general marasmus; but is especially seen in those who have died of cancer, when the heart is found of very small size. Thus, in a recent case of cancer of the stomach, the heart weighed only four ounces. We do not think there is any obscure connection of this atrophy with cancer; but the wasting of the heart is proportioned to the wasting of other parts; whereas, in most other wasting diseases, especially in phthisis, the implication of the lung induces a resistance to the flow of blood, and a consequent comparative hypertrophy, so that the heart generally has its normal weight. If any part of the heart receive a deficient supply of blood it wastes; and thus, when the mitral orifice is small, the left ventricle is small, unless some other cause of enlargement is present.

Sometimes the organ wastes if there is much external fat. Such hearts do not look small. The subjects of the change die suddenly, and not slowly by dropsy. But in this interesting specimen you will see the heart very small and thin-walled. It weighed but five and a half ounces. The thin walls are chiefly composed of fat. It came from a woman who died of gradual cardiac dropsy, such as results from common obstructive heart disease. We look on this case as a very rare example of fatty atrophy of the heart. It has been said that fatty heart leads to dropsy and the other conditions of cardiac obstruction; but that statement does not accord with our experience.

Indeed this is the only example of dropsy from fatty disease of the heart that we have ever seen.

Hypertrophy of the Heart.—Simple hypertrophy of the heart is not often found post-mortem, except in one disease, in which it is frequent enough. This disease is granular kidney. The heart, in granular kidney cases, is *nearly* always enlarged; it may reach eighteen to twenty-four ounces, or even more, but twenty ounces would be a considerable hypertrophy. In such cases there may be no dilatation, but a simple hypertrophy. We must not say with Rindfleisch that hypertrophy is invariably associated with dilatation; a twenty-ounce heart may have no cavity at all after death. But we would impress on you that all examples of hypertrophy *due to valvular or otherwise primary cardiac disease* are always associated with dilatation. This constitutes a distinction of great importance, because dilatation lowers the efficacy of the hypertrophy, increasing as it does the surface which the stronger muscle has to operate upon in contracting. Niemeyer, in looking for changes in the muscle to explain dropsy in simple dilatation, appears to forget this fact. From many careful observations we are convinced that the symptoms ascribed to hypertrophy are due to *dilated* hypertrophy, and are severe in proportion to the dilatation. Patients with granular kidney and simple hypertrophy are not aware of any change in the heart. They are always adult, and often their costal cartilages are so stiff, and their lungs so expanded, that the small increase of bulk which a simple hypertrophy causes is not to be discovered. As soon as dilatation sets in then begin the symptoms, and the trouble is in proportion to the dilatation. Simple hypertrophy does not cause symptoms and never exists as a primary cardiac disease.

In Bright's disease, so far as we have observed, hypertrophy is limited to the left ventricle, or nearly so limited. The right appears to share a little in the enlargement, but this is probably due to the fact that many of the fibres extend across both ventricles and no doubt must hypertrophy in their whole length, so as to thicken both ventricles. The convexity of the enlarged left ventricle often encroaches on the right. The hypertrophy in Bright's disease is probably due to resistance to the passage of blood through the vessels of the general system, and the excess of such resistance may at last lead to dilatation of the heart. The renal symptoms are then complicated with and soon outweighed by the cardiac.

Dilatation with Hypertrophy.—It is usual to arrange the various forms and varieties of hypertrophy and dilatation as follows: *hypertrophy* is spoken of as *simple* when there is a mere addition of muscle to the exterior of the walls; as *concentric* when it is at the expense of the interior; and as *eccentric* when the cavity is enlarged. As regards *dilatation* there are that which is called *simple dilatation*, *dilatation with hypertrophy* of the walls, and *dilatation with thinning* of the walls. You may keep these six terms in mind, since they are useful in examinations, but they are, for the most part, of exceedingly little importance and must have arisen in some author's study rather than in the course of inspection of the state of the heart after death.

The terms active and passive dilatation have been used: passive dilatation meaning the giving way of the ventricle, and, therefore, signifying thinness combined with dilatation; active dilatation, meaning dilatation with thickening. There is no evidence, however, that the heart dilates other than passively, so that the ground of the distinction is not good.

The terms active and passive aneurism have also been applied, but these had better be avoided. For, besides the objection to the word active, there are, as we shall see, true sacculated aneurisms formed from the heart, and for these the word aneurism must be reserved.

The six different forms of enlargement just enumerated can easily be reduced to four, for it will be seen that two are identical—the eccentric hypertrophy and dilatation with hypertrophy; and it is more than doubtful whether there is such a condition as concentric hypertrophy, so that this also may be struck off the list. The cases of supposed *concentric hypertrophy* have generally occurred in persons who have died of granular kidney or of thickened right heart in cases of malformation; here the heart is found empty, and the ventricle firmly contracted on itself; the cavity being thus obliterated an appearance of hypertrophy is given to it, which would suggest the increase of its thickness by growth of fibres inwards as the name infers; but if such a heart be kept for some time it will relax, and a cavity be formed in it as usual. Thus, so far as the hypertrophy is concentric it merely shows a mode of death. Hence, with all the respect due to the authority of Rokitansky, Bamberger, and some others, we believe there is no concentric hypertrophy.

As to the four other conditions—we have already spoken of simple hypertrophy—those that remain are simple dilatation, dilatation with thickening, and dilatation with thinning. But we believe that all morbidly dilated hearts are invariably hypertrophied. This is shown by the increase in weight they always show. It is easy to mistake the soft open state of a heart in its diastole for a morbid dilatation, and we think that the condition called "simple dilatation" is nothing more than this state of diastole; so that it is not a sign of disease in the heart but an accident of the mode of death. It is the opposite of concentric hypertrophy, which we have said is the state of systole persistent after death, while simple dilatation is the state of diastole persistent after death.

Thus, then, simple hypertrophy has no symptoms, and concentric hypertrophy and simple dilatation are accidents of the mode of death and not heart disease at all, and eccentric hypertrophy and dilatation with hypertrophy are the same thing. There remain but two conditions out of the six, viz., *dilatation with thickening* and *dilatation with thinning* of the heart's wall. Now, we repeat that we never have known morbid dilatation (we mean dilatation with conditions and symptoms referable to heart disease) without increase in the weight of the heart, or in other words hypertrophy of the heart. And so these two conditions are both of them examples of dilatation with hypertrophy; but the distinction between the two is really very important. For when dilatation is great in proportion to hypertrophy, then the danger and distress are proportionately great. So, indeed, that this rule will be found true;—cardiac disease is dangerous in proportion as dilatation is greater than hypertrophy. For the greater the hypertrophy the greater the power to carry on the circulation and the less the tendency to death. Thus, *dilatation with thickening* indicates in the living a tendency to longer life, and in the dead it indicates that the struggle of the heart against its difficulties was longer and better maintained, while *dilatation with thinning* indicates the reverse of all this both in the living and the dead. In distinguishing the two conditions, we must, however, remember that they of course graduate into each other, so that sometimes you find dilated hearts with the walls of a natural thickness, and neither thicker nor thinner. Some use the term "simple dilatation" for this; but such use of the term spoils the whole view of the conditions under consideration. There is no significance whatever in the maintenance of a natural thickness in the wall during dilatation, because any dilatation requires an increased thickness of the wall to restore the balance between the power of the heart and the resistance of its contents; so that it is of no use whatever to see that the wall of a dilated heart is of natural thickness, much less to give a name for this observation. Such a name for simple dilatation is simply misleading. What proportion of hypertrophy is required to overcome each degree of dilatation is an almost insoluble problem; but it is certain that the hypertrophy needs to increase very rapidly, and that danger is in proportion to the insufficiency of the hypertrophy. This insufficiency may be due to suddenness in the production of obstruction or regurgitation at one of the orifices. But it is generally accompanied by a degeneration of the muscular tissue, or it indicates some constitutional weakness on the part of the patient, by reason of which the chambers of the heart give way when over-distended.

When the left ventricle is much stretched, we said the term aneurism had been used; but, though we have avoided it, yet in some instances we can scarcely adopt any other name, as in this example, where you see the left ventricle immensely distended, and the walls thinned into a membrane scarcely thicker than a bladder. The analogy with aneurism is generally maintained in these cases by the occurrence of ante-mortem coagulation of blood in the dilated cavity; but the state differs from aneurism in this essential point, that it does not produce pressure as a tumour.

In hypertrophy without dilatation, or with little dilatation, the muscular walls may be uniformly thickened, and the trabeculæ or columns within participate; this is remarkably seen on the right side, where these muscular bands are often of great thickness, especially those columns that connect the free wall of the right heart with the septum, called by Mr Wilkinson King "moderator bands," from their function in restraining excessive dilatation. In the left ventricle the thickness is generally of natural proportions when the hypertrophy is without much dilatation; but if combined with dilatation, the walls are thicker at the base, and become gradually thinner towards the apex. In these cases the mitral fleshy columns are not correspondingly enlarged, but have a tendency to become narrower or pointed towards their upper extremity, and to change to fibrous tissue at their summit. It appears that this fibrous change of the musculi papillares has a conservative tendency, for if the cavity of the ventricle be dilated, the tendinous cords of the mitral cannot reach across it unless they are elongated in proportion to the increase of the heart's diameter, so that the valve-flaps in such dilatation are drawn down in the systole instead of moving across the mitral orifice. Such stretching down of the mitral columns and cords is a common cause of mitral regurgitation in dilated hearts, notwithstanding that the valves and cords may enlarge and elongate to some extent to suit the new conditions. Indeed, one may say that all *greatly* dilated hearts always allow mitral regurgitation in consequence of insufficiency in the length of the mitral tendons. It is plain, therefore, that the change of the column to fibrous tissue, and consequent persistence of its length during systole, has a conservative tendency. But when the overstretched mitral cords break, then the regurgitation becomes much more severe and irrecoverable, the broken cords get covered with fibrin, and the friction of this extends the mischief. Thus arise most of the cases in which mitral disease complicates dilated hypertrophy of the left ventricle.

The altered conditions of the cavities of the heart, then, are three: a simple thickening of the walls, a dilatation with thickening, and a dilatation with thinning. These conditions are found in all parts of the heart, but are especially noticeable in the left ventricle. The right ventricle being usually simply thickened or dilated with thickening; the right auricle generally dilated with slight thickening; while the left auricle is considerably hypertrophied with its dilatation.

As to the form of the heart the three most usual changes are as follows :—*Firstly*. Enlargement of the right side in emphysema (we distinguish this from simple wasting of the lungs in which the heart is small) or cirrhosis of the lungs, in either of which diseases the capacity of the pulmonary substance for blood is narrowed so that the right heart widens and enlarges from inability to empty itself, while the left remains small or even shrinks, always shortening a good deal, so that the right heart coming in front of and below it forms the apex of the whole heart. Thus, the organ is broad; or, in fact, has more or less a rounded form instead of being conical. It is important to recognise this, for it tells a tale of long-standing pulmonary obstruction. In emphysema, through the large lungs depressing it, the heart lies more in the middle line of the chest, and lower down than when the left heart is enlarged. In cirrhosis of the lung it is displaced towards the diseased lung.

Secondly. In cases of contraction of the *mitral orifice* the figure of the heart is at first sight much the same as that just described, for the obstruction soon is felt through the gorged lung in the right side of the heart, but the left auricle first becomes overcharged, consequently distended, and its walls hypertrophied, so as to reach often a very great size, while its walls become remarkably thick and tough, and uniformly so throughout; the dilated left auricle presses on the left bronchus, as was pointed out by Dr Barlow, so that signs of the pressure may be detected with the stethoscope. Thus, the heart in contracted mitral orifice is rounded from enlargement of right side, and with this there is an enlarged left auricle. In simple obstruction of the auriculo-ventricular orifice, the left ventricle is found smaller than natural, but often some other diseased condition, casually associated, gives rise to its enlargement.

Thirdly. In *aortic disease* we have immensely enlarged or *bovine* hearts, as they are sometimes called. Owing to the obstruction at the mouth of the aortic orifice, and the subsequent reflux of the blood, the left ventricle becomes often immensely distended, and its walls thickened. In young subjects the large heart may then obviously bulge out the left chest, the intercostal spaces over the heart being widened. The left ventricle elongates, so that the heart is longer, and it extends further downwards and to the left; we do not find its axis more transverse through gravitation as some say; the connection with the trachea prevents this. If there be much aortic regurgitation, and the muscle be poor in texture, the ventricle is often much thinner at its apex than at its base. All the other parts participate in their turn in the consequences of this impediment to the flow of blood through the left ventricle; the left auricle becomes unable to empty itself, and the effect of the obstruction extends round to the right side, as just now mentioned, so that the whole heart becomes enlarged.

Fourthly. Besides these examples of enlargement from obvious causes, we meet with cases of *simple enlargement* without any valvular disease, and these constitute, generally, the largest hearts found in museums; an example is here seen. In some of these the orifices are increased in size, but this may be a consequence of the ventricular distension. Thus the left auriculo-ventricular orifice is enlarged, and probably allows the blood to regurgitate, for the ventricle is often greatly distended at its posterior part behind the valve. The frequent association of this simple form of enlargement with Bright's disease and rigid arteries suggests whether it be only a further stage of the ventricular hypertrophy already mentioned, and that, owing to the obstruction being greater than the hypertrophy is able to overcome, the hypertrophied left ventricle cannot empty itself, and dilates, while its gorged condition obstructs the lung and right heart until the usual hypertrophic effects are brought about in these. Dr Gairdner thinks such enlargement may be due in some cases to a contraction of the lung from old pleurisy, so that the lung cannot fill the chest, and hence the heart expands through the disproportionate suction exerted on it in the act of inspiration.

Lastly. We have met not unfrequently with cases which have convinced us that dilated hypertrophy of the right heart and pulmonary arteries must be distinguished as an important and idiopathic disease. Sometimes the right heart is of enormous size, and the pulmonary arteries dilated so that the third division of the branching produces arteries as large as or larger than the normal main trunk, while the main trunk itself is two inches in diameter. The small arteries throughout the lung stick out on section like crow-quills. We have in such cases searched vainly for any condition of lung to explain the dilatation of the heart, and are obliged to conclude that it is idiopathic.

It may be as well to mention here, in connection with hypertrophy, the changes which take place in the muscle. The question has long been asked, do the muscular fibrillæ grow larger in hypertrophy, or are new ones formed? There are some who think that the fibres actually grow in size, but of this there is some doubt, although there can be none as to their increase in number. We have often examined hypertrophied hearts, and have found great difficulty in making out the points in question; but we have seen nucleated fibres which probably were the germs of new tissues. The hypertrophy of the right ventricle differs remarkably from the left: if you feel the two ventricles of this hypertrophied heart you will perceive that the left can be readily torn through by the finger, while you cannot do this to the right without considerable difficulty. Why this tough leathery consistence should occur on the right side and not on the left is not altogether clear; whether the original texture on the two sides is in any way different, or whether there is any condition of the mechanism of the right ventricle which may account for it, we have not yet determined. It is important that you should recognise this change of consistence in the right heart, for thus you will be able to ascertain the hypertrophy when the thickening is scarcely visible.

Obesity of the Heart.—This is met with mostly in old people who have grown fat, and then it forms part of a general fatness, bringing with it, however, special dangers. Sometimes it is found in persons who are not stout, yet have a greasy atrophic look; a condition of greasy leanness in elderly people is worthy of your attention, because it is thus apt to be associated with fat heart, and the same disposition is seen in those who have been intemperate, especially those accustomed to an excessive use of malt liquor. In such we may find the front of the heart wholly covered with fat, so that only a small portion of the muscle of the right ventricle can be seen; and, on cutting into the cavity, we find that the adipose tissue has penetrated the muscle and streaked it throughout, or, by its encroachments, it may have actually destroyed it, especially in a part of the wall of the right ventricle. This is generally at the apex, but sometimes at the base also. You then find that the whole thickness of this part of the right ventricle is made up of fat, and you can see by looking at the part closely where the layers of muscle run into and lose themselves in the fat. Under these circumstances the muscular fibres generally simply waste away by the pressure of the fat. Examining sections by low microscopic powers you see the fat growing in the course of the vessels; you can see this also plainly on the surface with the unaided eye.

The whole right heart may be covered with fat. On one occasion it reached the thickness of half an inch, the thinnest part being one third of an inch thick over the middle of the right ventricle; but this quantity of fat is very rare. We have never seen the left ventricle covered all over with fat. Obesity is sometimes associated with great atrophy of the heart. Thus, a heart thickly covered with fat may weigh only seven ounces.

True or Saccular Aneurism of the Heart is that form of disease where there is a sacculated pouch from one of its cavities. The most marked cases are those where the pouch is circumscribed and communicates by a small opening with the larger chamber within; in these instances there has generally been no history of acute inflammation of the heart, and therefore there is no proof that they result from such a process; they are found often towards the apex of the organ, while the acute aneurisms we shall speak of under acute endocarditis arise mostly in the neighbourhood of the valves. Those in question seem rather due to a chronic process, and are formed like aneurisms in the blood-vessels, or still more like the saccular pouches of a hypertrophied bladder, which are called "herniæ" of its mucous membrane. The heart, like the bladder, shows an uneven surface within, areolated with columnæ carneæ. The spaces between these are weaker parts of the wall. Such a weak space may yield when the heart is a little dilated, forming a pouch at first in the wall of the heart, which as it increases projects on its outer surface, yet, even if large, it still communicates with the ventricle only by a small hole representing the little area between the columns which originally yielded. So far the formation of the saccular aneurism of the heart is exactly like the sacculation of the bladder-wall, and a degree of such sacculation is not unfrequent in dilatation, favouring ante-mortem coagulation. But a fibrous degeneration appears early in the wall of any large pouch. Some say this is the first step; it may be so in cases of more general yielding, where the sac opens by a wide mouth into the ventricle, but in the more typical kind of cases we are now speaking of, where the sac communicates by a small hole with the heart, we believe the process begins like sacculation of the bladder, and the fibrous and calcareous degeneration follows afterwards. These pouches are mostly found at the apex of the left ventricle, though sometimes in the auricle, especially the left; the ventricle is itself enlarged and atrophied, with the thin-walled sac coming off from it. You will see the condition in this specimen, and also in this, where the aneurismal dilatation burst. One of our best specimens is this, which came from a patient who died of phthisis. It was found accidentally, and had undergone changes which had resulted in a cure; it is situated, as you see, at the apex of the heart, and was firmly united to the pericardial sac. It is about the size of a pigeon's egg, of an oval shape, its walls bony and very hard, and communicating with the ventricle by a small opening which would only admit a quill. Its interior was occupied by a soft translucent matter, which appeared to be fibrous, and had undergone considerable degeneration. We met recently with another example exactly like this, in the possession of Mr Townsend; the aneurism had undergone complete cure.

Injury and Spontaneous Rupture of the Heart.—Although two distinct conditions are here referred to, we place them together in order that they may be contrasted, as their recognition is of great practical importance. The one is rupture of the heart from direct violence, and the other that which arises spontaneously; the correct knowledge of the characters of spontaneous rupture may be of the greatest consequence in a trial for manslaughter. Spontaneous rupture of the heart, in nearly every case, arises from a softening of the tissue, and may be considered, therefore, as indicating generally a diseased organ. But in this specimen, taken from a brewer's drayman who died suddenly while lifting a beer barrel, and in whose left ventricle there is at the back near the apex a rent two and a half inches long, we cannot discover any important change in the muscular substance of the spirit-kept preparation. Spontaneous rupture occurs, for the most part, in the left ventricle. The other parts, especially the right ventricle and auricle, are less frequently affected; whereas the part of the heart most liable to injury by violence is the right ventricle, or front part of the heart, as you might expect. Other parts may, however, suffer. Thus, we had a case of traumatic rupture of the left auricle just below and internal to the apex from a fall in which the sternum was not broken. Rupture from disease occurs mostly in persons advanced in age, in whom a fatty degeneration has taken place; and the laceration occurs generally in the left ventricle, towards its apex, by a slit more or less transverse running in the course of the fibres, and sometimes an inch or two long; in some exceptional cases the rent has reached the septum. Sometimes the laceration, from either cause, is merely on the exterior of the heart, and has not penetrated the interior, as seen in this specimen, where two or three superficial rents are seen, and death occurred from hæmorrhage into the pericardium. Sometimes, as in this specimen, the laceration is limited to the inner layers.

The differences we have just enumerated enable us to form a nearly certain judgment in cases of rupture of the heart as to its spontaneous or traumatic origin. Most examples of traumatic rupture are parts of some very severe injury which is otherwise manifest enough, for the healthy heart is not ruptured except by extreme violence. The most stable grounds of conclusion in spontaneous rupture are drawn from the heart itself, while the conclusion is rather drawn from the coexistent conditions in traumatic rupture, for the rent through injury *may* happen in almost any part of the organ, although less likely to occur in the left ventricle, *i. e.*, the part most liable to spontaneous rupture. In speaking of the degenerations of the heart we shall show some reasons why the left heart suffers most in all spontaneous disease. We have met with some cases which proved that spontaneous rupture of the heart had occurred at two stages with at least several weeks' interval.

Punctured and Gunshot Wounds.—These are by no means invariably at once fatal, patients living sometimes for weeks after such injuries, while slight wounds may probably be recovered from. The injury, if not fatal at once, produces death by the subsequent inflammatory results. In this specimen, which was given us by Mr Callaway, the heart is traversed by two bullets; but the man spoke after the receipt of the wounds. Many of you saw a patient who was in the hospital, who was stabbed through the pericardium, although the heart appeared untouched: the man lived two days. A curious case was recently shown at the Clinical Society, where a needle, which had been driven into the chest, moved with the action of the heart so as to convince the observers that the needle was in the heart itself.

Effusion of Blood is sometimes met with in the substance of the heart, in cases of purpura, or other diseases where the blood is affected. Also beneath the endocardium, on the interior, such purpuric patches may be seen; and this has long been noticed as occurring in cases of poisoning by arsenic and mercury, and especially by phosphorus. In cases we have examined in this hospital after deaths caused by all these kinds of poisoning, patches of ecchymosis were found in the right auricle and left ventricle.

CHANGES IN THE MUSCULAR FIBRES

Passive Changes.—*Softening.*—The heart is found soft when its *rigor mortis* has passed off, as you know from your experience in the dissecting-room, where the heart is generally almost pulpy by the time you reach it in the usual course of dissection. You will perhaps remember that at the same period the spleen is very pulpy, and the vessels contain air bubbles, while their interior is deep red or purple from imbibition of serum stained red through solution of the corpuscles in it. These are phenomena that accompany decomposition, as you well know. Now, in some cases the softness of the heart is very remarkable when there is no sign of decomposition about the body generally. When this is the case you will find the spleen more or less pulpy, and the vessels deep red within, and containing air just as in advancing decomposition. The course of the vessels under the skin is then marked by purple lines, and the trachea and bronchi are stained intensely red. We have known this change supervene in five hours after death, and that in winter time, when the weather was cold, and when no signs of decomposition appeared in other bodies kept for several days. By considering the kinds of cases in which this early decomposition of the blood and softness of the heart and spleen appear, you will find that it is more common in fevers, in persons who have died obscurely in two or three days after great surgical operations, or in persons who have died by coma in jaundice; that is, in short, where there are other reasons to believe that death was caused by changes in the blood. Speaking generally, we should say that marked softness of the heart is an index of changes in the blood, and goes with pulpy spleen, red endarterium, and at last air bubbles in the vessels.

Such pulpy softness of heart is of great importance among the signs of blood changes; so that when it supervenes very early after death, when the rest of the body is free from decomposition, there is great probability that the unusual condition of the blood thus declaring itself was in progress during life—we would not say to the extent of forming air within the vessels, but this may happen. We have had occasional evidence that emphysema from decomposition, *without any gangrene*, may be found commencing during life, and the same gaseous exhalation may continue after death. In these cases the softness of heart, &c., may come on exceedingly quickly, being complete in a few hours.

But we have no knowledge of the state of softening of the heart to which some authors on the heart devote a chapter, ascribing special symptoms to it. Softness of the heart is important, but its importance is indirect as an evidence of febrile blood changes—blood solution (blutlösung).

Pigmentary degeneration.—The heart is sometimes brown and friable, having less consistence than is natural. The fibres show yellowish-brown pigment grains, which at first are seen about the nuclei of the fibres, and afterwards in streaks within the fibres. This condition is, according to our experience, very frequent, and is not a sign of any primary or special disease of the heart belonging to senile states and general emaciation from any cause. We would mention that this brown change is not found in the voluntary muscles.

Fatty degeneration.—By this we do not mean obesity of the heart (see p. 114), but a change in its muscular fibrils, such that their interior becomes loaded with fat, which obscures their striation until perhaps the fibres look like columnar aggregations of little bright round granules. This change may exist without any increase of external fat, and may occur in a person otherwise thin; it is also found often accompanying valvular disease of the heart. It may present either of two kinds of appearances—one where the whole muscle is affected; and the other where the inner surface of the heart is marked in a peculiar manner. In the latter condition the rest of the muscle is often of a good colour, and the external part healthy, but within the ventricle the heart is seen to have a peculiar streaked appearance; on looking carefully at it a number of white transverse zigzag lines are seen running parallel to one another, producing a feathery appearance, or rather they look not unlike the markings on a tabby cat. They are seen especially, and in the first instance, on the mitral columns of the left ventricle; afterwards they appear on other parts of the cavity, and in extreme cases may be seen in the right ventricle. We have never seen this change in the auricle. It only penetrates a slight depth in the substance of the heart. The question arises how long is such a process in formation, and what are its exact pathological indications? It is found in connection with other disease of the heart, and is especially well developed and obvious in those cases of simple anæmia which have been called idiopathic for want of other explanation. This condition cannot be shown in our museum preparations, for it altogether disappears after the organ has been kept a short time in spirit; but you will, no doubt, soon be able to see it in a recent heart.

To understand why the inner layers should especially suffer, we must remember that the innermost layers of the heart are furthest from the coronary arteries, and they are broken up into *columnæ carneæ*, a condition which limits the directions of vascular supply to the attached ends of the columns, so that one perceives that these inner layers are under comparative difficulties in obtaining nourishment, which may explain the frequency with which this fatty degeneration occurs in and is limited to them.

In the more general form which affects the whole thickness of the heart the muscle is found yellow-looking, flabby, and easily lacerable. This is the form of fatty heart which has long been known to be often associated with disease of the coronary arteries as a cause of *angina pectoris*. A good example of it is here. Sometimes a limited part, usually towards the apex of the left ventricle, is much more affected than the rest; it may be found, indeed, breaking down. The branch of the coronary artery going to this part is then generally more diseased than the rest; indeed, some cases have occurred where such a localised fatty patch, with its coronary artery very badly diseased, either by atheroma or embolism, has been found in a heart that was sound in other parts. Such a condition, combining power in the whole heart with weakness of a limited spot, strongly predisposes to spontaneous rupture at that weak spot; while a heart, whose ventricle is everywhere equally fatty, though less liable to burst, makes up for this by its tendency to stop suddenly.

Some surprise was created by the observation of Dr Hermann Weber, who stated that on chemical examination, hearts in this condition present no increase in the quantity of fat present. The amount of fat is certainly very small as compared with the amount in fatty degeneration of the liver and other organs. In an analysis kindly made for us by Dr Stevenson five per cent. of the recent muscle was found to be composed of fat, which is about twice the quantity present in heart-muscle that is microscopically healthy. This result agrees with those obtained in an elaborate series of researches by Krylow, which showed that although the amount of fat is always small in the change in question (four per cent. of the fresh, or twenty per cent. of the dry muscle), yet there is relatively a decided increase. Nevertheless, the amount of increase is positively so slight as to make it probable that the granular appearance of the muscular fibres is due rather to a rearrangement of their elements than to a large addition of fat, as was supposed before Dr Weber's researches were published.

Active Changes.—*Inflammation; diffused myocarditis of low degree.*—It is highly probable that in those very common cases of inflammation of the exterior and interior of the heart accompanying rheumatic fever the muscular structure itself is often involved. We think so for many reasons; first, in hearts with pericarditis and endocarditis we have met with proof, many years afterwards, that the muscle had been affected, in the fact of this structure being occupied by an adventitious fibrous tissue; secondly, the enlargements which occur after such inflammations, with only slight or with no valvular disease, cannot be accounted for except by supposing some altered condition of the muscle; thirdly, in fatal pericarditis, if the adjoining muscular tissue be examined, the fibrillæ will be found fatty; fourthly, we have clinically observed the heart to enlarge and its sounds and beat to become almost imperceptible during the course of rheumatic fever in some cases; and, lastly, it is only on the supposition of some alteration in the heart that we can account for some sudden deaths which occur in rheumatism. It is not remarkable that in cases of speedy death no very marked changes beyond softening should be observed in the muscle; and we think, nevertheless, that there is such an affectiou as an *acute general inflammation* of the muscle.

Ulcerative myocarditis.—This is sometimes met with in connection with endocarditis, especially when this attacks a valve; and, owing to the changes which generally follow in the substance of the heart, the name of *acute* or *false aneurism* has been given to it. The manner of attack is generally in one of the following ways:—An endocarditis affecting, as usual, the valves, reaches such intensity that the tissue of the valve ulcerates, and the ulcer spreads to the root of the valve and invades the muscle; or a great mass of vegetation attached to a valve by its direct pressure cuts into the muscle, forming an ulcer; or, what is more frequent, a mass of fibrinous clot attached to the moveable part of a valve, and, swinging in the blood current, comes sharply into contact with the heart's wall, and so by friction starts an ulcer; the muscular tissue becomes involved, and the ulceration spreads in the muscle more or less rapidly, so that an excavation is formed generally near the root of a valve.

However formed, this excavation of course communicates with the ventricle, and its contents and produce mix with the blood, so producing very terrible consequences. For, carried in the stream, these products, if the ulcer is intense, set up abscesses in remote parts, as the brain and liver; or by acutely inflaming and destroying the wall of a considerable artery, cause acute aneurism of it. But if the ulcer is of lower intensity, it gives rise to plastic inflammation in the parts where the "emboli," as such transported matters are called, lodge.

When examining the ulcer microscopically you see the muscular tissue of the heart sometimes only exposing a ragged surface with little infiltration of the substance; but sometimes the floor of the ulcer is charged with pus to a depth extending through the whole thickness of the left ventricle. We found in one such case portions of muscular tissue still plainly recognisable, adhering to the surface of the ulcer so lightly that a few more beats of the heart must have thrown *some of its muscle* into the brain or elsewhere.

When such ulcers are present in the heart you will always find larger masses of friable, half degenerated fibrinous clots, perhaps partly calcified, forming masses on the valves near the ulcer, and you will nearly always find that these masses of fibrin are so placed as to give rise to the ulcer by friction.

The ulcerative process may proceed outward, the visceral pericardium then is softened, and an acute pericarditis is set up, as seen in this preparation, from a case which occurred only a short time ago in the hospital. The patient was a young girl who had acute endocarditis; and you see the muscle was involved close to the outer aortic valve. The bursting of such a sac might possibly cause sudden death by hæmorrhage into the pericardium. The softening or abscess need not necessarily take its course outwards in this direction; for sometimes it proceeds between the valves, and forms a small aneurism there; sometimes such an aneurism may eventually burst upwards, between the ventricle and auricle; or, if the process be towards the septum, a pouch may be there formed, or an actual perforation; as in this case, where the acute ulcer at the root of the aortic valves had pierced through into the *conus arteriosus* close to the pulmonary valves.

Endocarditis in this acute and grave form constitutes a disease which is totally different in its history from the obstructive form of heart disease. The sufferer dies generally without dropsy often suddenly, and the case is marked by its subfebrile course and the occurrence of paralysis, aneurism, or the other evidences of embolism. Rheumatic fever is one common cause of ulcerative myocarditis; but the majority of the cases occur in young persons, generally males, under thirty years, and often under twenty-five. In most of these cases there has been no history of rheumatism

As usual in the heart diseases of extra-uterine life, the left side, *i. e.*, the hardworked side, is that which suffers most; but we have met with it on the right side, and a few other right side cases have occurred. The disease is more apt to extend from the aortic than the mitral valve. In two cases the illness was ascribed by the sufferers to excessive drinking; but these men had been soldiers, and it is known that military exercise overstrains the heart. It has appeared that overstrain has predisposed to and rheumatism or alcohol excited this grave disease.

Abscess of the heart.—This arises under conditions quite different from those which produce ulcer. A few cases are recorded of spontaneous suppuration diffused in the tissue of the heart; but we have never met with this. Abscess of the heart is practically always pyæmic, and it is seldom met with except in cases of violent and general pyæmia. We have seen several cases, and they have mostly been in youths who had suppurative periostitis, or acute necrosis of the long bones, where the kidneys also contained purulent deposits. The immediate cause of death in these instances is often a pericarditis from the bursting of small abscesses into the pericardium. You see in this specimen, besides the lymph covering the serous membrane, a number of small points of purulent matter within the substance of the heart itself. The character of these is peculiar, and resembles similar deposits in other parts, in their being surrounded by a halo of congestion, and which seems to indicate, as we shall hereafter mention, that this form of inflammation arises primarily from obstruction of the blood-vessels by emboli, due to the presence in the blood of some of the elements of pus; for in other parts of the muscle you could see, when it was quite fresh, small red patches of congestion, constituting the stage prior to the inflammation, just as usual in embolisms.

Sometimes these abscesses will burst into the interior of the heart, then the blood enters and washes out the pus which may be carried in the blood stream, producing embolism elsewhere. But it does not necessarily follow that any embolisms will then appear; for we have seen a case where a considerable abscess had burst into the left ventricle close to the mitral valve, and yet no signs of embolism were present in the body. It is said that abscesses in the wall of the heart may wither and become calcareous; but we have never seen any proof of this.

Subinflammatory or fibroid degenerations.—We have just said that the results of inflammation were seen in streaks of fibrous structure running into the muscular substance. Extreme examples of this kind are always combined with indications of previous endocarditis and pericarditis, and are allied to the changes seen in the liver, lungs, &c., where the investing capsule is the subject of chronic inflammation, which involves the adjacent texture, and thus, the pericardium being thickened and adherent, the neighbouring muscular walls become involved in the fibrous change. This is shown by a section of the wall of the ventricle, as in these specimens, where fibrous bands and streaks will be seen pervading the tissue in all directions. When these fibrous bands are seen along with obvious signs of old pericarditis or endocarditis, they are referred to the extension of these processes into the tissue beneath; but sometimes we meet with patches of fibrous substance in the tissue of the heart, when there is no evidence of former pericarditis or of endocarditis, other than the sharing of the inner surface in the change at the affected spots. The interior of the ventricle is marked with patches of a pearly whiteness, owing to a fibrous change of the endocardium, and the muscular trabeculæ beneath, when cut asunder, are seen to have a mere trace of muscular fibre in their midst, some of the smaller being wholly fibrous.

Such examples are not very uncommon, and their pathology is very interesting. Is it inflammatory? You sometimes find excess of new corpuscles in the growing edge of the patches and hence might infer that they are inflammatory. In one case we met with such tissue in the heart along with endarteritis which had led to closing up of several of the principal arteries of the body. This would infer a general inflammation of the lining of the vascular system. But often they histologically appear only as fibrous tissue, and the existence of former inflammation is an inference unsupported by history of rheumatism, &c., which in our cases has generally not been present; though this experience differs from that of some other observers.

Such patches are often clearly traceable to syphilis; we have before spoken (p. 94) of the liability of the muscles to syphilitic inflammation. The heart and diaphragm are among the muscles most liable to this change.

Besides these circumscribed patches of fibroid degeneration, probably syphilitic or otherwise inflammatory, you find in the interior of greatly dilated hearts that a fibroid change invades the *columnæ carneæ*, especially in the left ventricles. These may be changed to fibrous tissue. Such a change is probably due to a stretching of them, drawing the middle part of them away from their vascular supply, which must enter at the ends. The change you will perceive is always begun in the middle of the muscular columns and at the apices of the *musculi papillares*, that is, at the points farthest from the entry of these vessels.

Syphilitic Myocarditis.—This is not infrequently found not only in the form of fibrous scar, as stated in the previous paragraph, but also in well-characterised gumma. Its characters do not, however, then differ from syphilitic gumma of ordinary muscles, which see (p. 94.)

Tubercle.—This has already been spoken of, as affecting the pericardium; it does not, as far as we know, penetrate the heart's muscle.

Morbid Growths.—*Cancer* usually occurs in the heart under one of two distinct conditions, that is, either as an extension from primary cancer around, or as a secondary growth. The first of these conditions is most common; the heart is implicated in a mediastinal cancer, which grows into the pericardium along the veins; for these are generally implicated, while the arteries resist the disease; hence the auricles are invaded rather than the ventricles. The extent of the cancer of the heart in these cases is generally not large, but we have seen nearly the whole of both auricles much involved; and in one case, where, however, the primary disease was in the cervical glands and we could not be sure whether the heart was directly or secondarily invaded, almost the whole heart at first sight seemed covered with cancer. Sometimes, in our experience more rarely, though the experience of others differs here, the heart is affected with secondary formation of cancer, just as other parts might be. We have seen epithelial cancer of the skin of the neck associated with a secondary nodule in the right ventricle wall. In this case the inner face of the mass had on it two large polypoid cysts containing a clear brown fluid. The walls of these cysts were partly of fibrin and partly of cancer, and the latter ran continuously into the former so as to convince us that the fibrin was being changed to cancer. In the case of extensive cancer of the whole heart, above noticed, there were numerous ante-mortem fibrinous polypi, and microscopic sections showed cancer extending into them. We mention these cases in some detail because great doubt is thrown by good authorities on the growth of cancer in fibrin clot.

We have already noticed the occurrence of cancer nodules in the pericardium (p. 103).

The forms of malignant growth that we have found in the heart have been various. We have met *epithelioma*, *lymphoma*, *melanosis*, and *sarcoma*.

Among adventitious growths we may show you this heart, having two cysts on its front. They are formed of thin membrane, containing a fluid, and are traversed throughout by a number of delicate fibres. Of what nature these cysts are we cannot say. The heart came from a lunatic, who, while attempting to strike another man, fell down dead, and the cause was found to be a rupture of these cysts into the pericardium.

We must also show you this very remarkable specimen of growth upon the heart, an osteochondromatous tumour, like a large mass of coral, growing upon the organ, and as large as itself. Whether this is a growth secondary to similar tumours in other parts of the body to which it belonged, or whether it be the remains of an included ovum which had become attached to the heart, we cannot say.

Here are two true polypous growths in the heart, although these are rare. We have only these two specimens to show, and both are growths in the left auricle springing from the septum near the fossa ovalis. In this old preparation you will see a polypus semitranslucent like a mass of size, which was injected through the coronary artery, although the vascularity is not well seen now. This other heart came from a woman who was in Charity Ward a few years ago under Dr Addison for hemiplegia; after death a growth was found in the left auricle, and nearly filling it; it is of an oval form, as you see, and grows from the auricular septum. It consisted of nucleated fibres, and might, therefore, be called fibrous, or fibro-plastic, and when freshly cut we remember that it was distinctly vascular.

Hydatid.—This occasionally occurs in the heart, though it is rare in the human subject. The cysts grow, probably, in the muscular substance, and then cause death by rupture or pressure. In this heart, from a girl who died in the hospital about two years ago, the hydatid cyst is seen growing in front of heart, between the auricle and ventricle, forming a cavity between the two. It is of about the size of an orange, and at the bottom of the jar you see the hydatids which came from it. The bursting of the cyst caused fatal pericarditis. In another case, the rupture occurred into the right ventricle of the heart. In one of our cases the hydatid was placed at the back of the heart over the auriculo-ventricular septum, and grew to the size of a large plum; its pressure had completely occluded the coronary sinus, so that the opposed faces of the sinus were inseparably united and sealed up. Drs Greenhow and Cayley, to whom this specimen was referred, showed that water thrown into the distal part of the sinus found its way into the right auricle by small openings, which were enlarged venæ Thebesii. We may mention that there are cases recorded of hydatid of the liver bursting into the pericardium.

The *trichina spiralis*, which infests most of the muscles of the body, and all the other striped ones, is never, or extremely rarely, found in the heart.

ENDOCARDIUM AND VALVES

Atrophy and Hypertrophy.—The endocardium is usually considered as corresponding to the internal coat of the arteries, but their pathological relations are not so much alike as this view of their nature might infer. Thus, if we exclude the valves from our consideration we may say that the endocardium does not suffer that fatty degeneration which is so very common in the lining of the arteries. Indeed, the endocardium is liable to but few changes, and in estimating those that it does offer we must bear in mind that it differs naturally in thickness in the different cavities of the heart. It is much thinner in the right side of the heart; indeed, in the right ventricle it can only be recognised by the polish its presence produces. On the left side its thickness is greater, especially in the left auricle, where it is naturally opaque and whitish.

Inflammation.—*Distribution of endocarditis.*—You will, we hope, have noticed that except obesity, which may not be disease, all the diseases of the heart are very far most frequent on its left side; indeed, endocarditis of the right side is exceedingly rare, except in cases of malformation. You will remember that we just now said the endocardium of the left heart is thicker than that of the right. Also, you may recall that in all we have told you about the diseases of the heart as distinguished from its malformations it was on the left side that the disease was most liable to occur. This is true of the fibroid patches, syphilitic or otherwise; also of the fatty changes, both that occurring in streaks and that permeating the whole thickness in the overworked hearts of aged people. Thus, we may say that the strongly acting and hard-worked ventricle suffers most disease. We shall go on presently to show you that the seats where endocarditis declares itself in the left side of the heart are almost invariably places where friction is exerted, and we shall have to point out that the rarer cases, where endocardial thickening and inflammation occur in the right heart, are always cases of unusual hypertrophy of it; where it exerts more mechanical power and, therefore, causes more wear and tear of the structures connected with it. All this evidence accumulates to prove that the reason why the left heart suffers more than the right is because it is stronger and works harder, straining the mechanism of its valves, and irritating its lining membrane with the friction of its blood upon it, while the right heart escapes because its play on its contents and valves is milder and less forcible. As a further proof of this, remember that in fœtal life, where the right heart is doing all the work, any disease almost always attacks the right side of the heart.

General characters of endocarditis.—Next please to note that when we speak of inflammation of the endocardium or endocarditis, you must not think that any such process ever occurs all over the endocardium, as pericarditis is found all over the pericardium, or peritonitis all over the peritoneum; that is, you never find the whole endocardium even of a single cavity coated with lymph or other products of inflammation. The inflammation is always circumscribed. You may occasionally find several such patches of endocarditis, discoverable by granulations, or fibrin on the membrane; but, except in the rarest cases, these patches are within reach of a fibrinous clot on a valve, which no doubt struck the affected part in the action of the heart; when considering the possibilities of such contact in any specimen, you must bear well in mind how the heart closes its cavity in contracting, and thus brings together parts that in the dilated and dead heart are remote from each other. The study of a great number of cases has led us to conclude that such friction, with fibrin clots, together with mechanical strain, make the principal if not the sole *direct* causes of endocarditis; rheumatism and other general states creating only a vulnerability of the fibrous structures, so that they cannot resist the irritation of the friction.

As a corollary from these facts, it follows that we must in all cases, where endocarditis is suspected, do all in our power to moderate the force of the heart's action, so as to place the left heart in much the same condition as the right, reducing the friction which we have nearly proved to be the sole efficient cause of the anatomical changes that result from endocarditis.

Comparison of endocarditis with other inflammations.—When inflammation was regarded as an act proper to the vessels, and when it was doubtful whether the endocardium had any vessels, it was naturally a rather perplexing question which inquired whether the apparently inflammatory effects of endocarditis were really due to inflammation. The difficulty was much greater in the case of the endocardium than in those of other evascular structures, such as the cornea or cartilage which produce little or no inflammatory products; because it was easy to deny the existence of inflammation in the apparent absence of its results. But a great quantity of lymph-like deposit was seen in endocarditis, and hence arose a rather keen discussion, some being disposed to think this disposal was really formed from the endocardium as lymph is produced by other inflamed serous surfaces, while others thought the apparent lymph was only fibrin of the blood, which had precipitated itself, especially as fibrin, was known to be in excess in rheumatism, the disease that commonly causes endocarditis. The following out of this question by Lee and others led to some interesting experiments on the inflammability of the inner vascular surfaces, and it was proved by them that the endarterium will not produce lymph and pus freely on its free surface like the ordinary serous membranes.

But the great number of more careful microscopic examinations which have been made of late have settled the question decisively, although with some alteration of the standpoint from which it is viewed. This alteration arises from the fact that inflammation is no longer regarded as *exclusively* the act of the vessels, although the actions in the vessels compose the chief and most obvious phenomena of ordinary inflammations. It was clearly shown by Virchow and others that a more constant, and perhaps the essential, act in inflammation is one of irritation in the parenchymatous parts of the texture as represented by their cellular elements. With this belief it has become no longer possible for any to hold the opinion that endocarditis cannot occur on the ground that the valves are evascular, while it is further proved that some at least of the valves, certainly the mitral, do contain capillary vessels which have been observed to be congested in inflammation.

Then at present we are able to follow out the inquiry into the process of endocarditis on the same terms as we examine the inflammation of other tissues, so far as the structure of the endocardium is concerned. But it remains true that its circumstances are very peculiar in that it exposes its large surface to contact with the blood. For the living blood is proved by observations and experiments to be always ready to deposit fibrin on a roughened surface, and especially so when the fibrin is in excess in the blood, and when the blood is arterial, and when the blood is in contact with a surface of no vitality, or low vitality, and when its current is checked. Now, all these conditions are found in the inequalities of an inflamed endocardial surface, and especially when the fibrinous deposit has already commenced on several adjacent spots. For there is then a rough surface of low vitality retarding the current in its inequalities; the blood from inflammation being hyperinotic, and the left heart containing arterial blood. Recognising these conditions, one is prepared to believe that any change in the endocardium, inducing a roughening of its surface by swelling or exudation, would soon lead to the deposit of concretions on the rough parts. Such a deposition undoubtedly occurs, and it is this lodgment of fibrin in quantities on the inflamed surface that constitutes the peculiarity of endocarditis, and causes it to differ from inflammation in all other parts. It is a most unfortunate thing that in the heart, where the consequences are so unhappy, the effects of inflammation are so permanent, and we may ask why it is that, when rheumatism affects the joints and the heart only equally severely, the former should so commonly recover their integrity, while the latter is permanently damaged. The permanence of the injury in the case of endocarditis is simply due to the want of counterpressure. In the joints the swollen membranes are pressed against the other solid structures as soon as the liquid effusion is removed. This pressure causes absorption of all the new products, whereas in the heart there is no direct pressure of solids against the inflamed valves, which stand freely in fluid blood, so that the new products persist.

Special characters of endocarditis.—Endocarditis is either plastic or ulcerative. Let us consider, first, the characters of its plastic form. When found in its earliest stages it appears as a slight swelling, with sometimes a pink colour of the membrane; as to this colour, we are not certain whether it can be regarded as due to congestion or imbibition of colouring matter. This always occurs near the edges of a valve in the formation of a line of little elevations along the contact line of its segments, where the friction is greatest. Some have thought that this is due to a peculiarity in the composition of this contact line of the valves. Now, it is true that this line is usually more fibrous and thick than the rest of the valve, but the fibrous thickness itself is clearly due to the chronic irritation of the line by the action of the heart. For (1) it is not found in young subjects and is thicker as age increases; and (2) it is not found in the valves of a normal right heart; (3) it is found in the valves of the right heart when that heart is hypertrophied. This line of little elevations is what we find in chorea, in acute rheumatism, in puerperal pyæmia, &c., that is, generally in acute plastic endocarditis, when the change in the heart is quite early. Such a change may give rise to a soft bruit in the action of the heart, but it cannot much obstruct its orifices, nor can it poison the blood with its products, so that at this stage it is of little importance. It is only its after consequences that are grave. We have already pointed to the absence of counter-pressure as causing permanence of the swelling of the valve; hence it is that the heart after acute rheumatism, chorea, &c., exerts its strain on an unrecovered, thickened, and softened structure. It is curious to observe how constantly we find in all cases of endocarditis from chorea, acute rheumatism, pyæmia, &c., that the change in the valves is limited to this line of bead-like elevations along the meeting edges of the segments. If a valve with these nodules be cut for the microscope across the plane of its curtain, so as to show a section down through one of the small nodules, this will be found to be composed of a simple cloudy swelling of the tissue of the valve through a multiplication of the cellular elements in its fibrous structure, which here and there by its excess raises the surface into a little hillock. If the hillock takes the form of a distinct projecting grain you will always find on the top of it a cap of fibrin separated from its substance by a line which the microscope defines very clearly. This cap of fibrin differs in composition from the hillock itself, though the difference is more easily seen than described, for the organization in both is very low; but the fibrin is almost structureless, while the hillock of swollen valve-substance shows the regularly placed nuclei of fibrous tissue.

Specimens of endocarditis in this early stage are frequent enough, but it is not easy to say what occurs next in the process, because we do not have many opportunities of seeing the intermediate conditions between this which is found in cases of death from the acute disease which causes the endocarditis, and that advanced state of change in the valves which long afterwards proves fatal by disabling them.

It appears to us that in the interval between acute endocarditis of rheumatic fever and the death long after from valvular disease of the heart, many frequent repetitions of the inflammation must occur; sometimes we find inflammatory products of two distinct dates on the valves, some recent, some older. But, more usually, a constant state of inflammatory irritation persists, slowly changing the valve. The cause is probably this—that the valve remains swollen through absence of the pressure on it which is required to restore it, as we have already said; and being thus unable to return to its proper size, while it is still softer than natural, it is both subjected to more friction and less able to resist this effect of the constant action of the heart; hence there is a chronic irritation of the unhealed valve, which gradually leads to great thickening of it, so that a scar-like tissue results, in which calcareous salts are often deposited, the whole causing those miserable effects in contraction and deformity with which we are all too familiar. Here, again, we would impress on you the necessity of warning all persons who have had rheumatic fever or chorea against such muscular exertion as will greatly increase the action of the heart and the friction of its valves.

The *ulcerative* form of acute endocarditis always begins in a valve. It must not be supposed to usually accompany the milder plastic form we have just described. Some authorities proceed in their description of endocarditis as if when the swelling of the membrane occurs the next thing usually is for this swelling to break down into an ulcer; but we must not let such an impression mislead us into thinking that ulcerative endocarditis is an ordinary part of common cases of endocarditis, such as we meet with in chorea, rheumatism, &c. The occurrence of ulceration is a rare and formidable complication of plastic endocarditis.

We have already described the process of ulceration in the muscular substance of the heart. When limited to the endocardium, *i. e.* when it is only in a valve, an abrasion of the inflamed surface forms and the affected side of the valve suffers a breach. The valve is, as you are aware, composed of two layers of endocardium with some fibrous tissue and a few vessels between them. Now, when one layer of the endocardium is breached by the ulcer the force of the heart drives the blood into the hollow and presses before it the remaining layer of the endocardium, thus forming an acute aneurism of the valve. Such an aneurism, of course, projects away in the direction of greatest pressure, so that in the mitral valve it bulges up into the auricle, in the aortic valves downward into the ventricle. It is but too easy then for the ulceration or the heart's action to work through the remaining layer of endocardium and so perforate the valve; such perforations are not very infrequent either in the mitral or aortic valves; they are always covered all around with "vegetations," *i. e.* nodular masses of fibrin which hide the opening. These vegetations may reach a large size and become calcareous, and by friction start ulceration in the wall of the heart where they come in contact with it. Such ulcers and perforations of the valves, with the ulcerations of the muscle of the heart which are apt to extend from them, constitute a dangerous disease; its characters are quite distinct from the plastic form of endocarditis, which is only dangerous through its subsequent effects in causing contraction of the valve and so inducing dropsy, &c. Ulcerative endocarditis is generally accompanied by pyrexia; it may produce pyæmic suppurations by embolism of distant organs with particles from the ulcer; or the large fibrinous masses around the ulcer may move off and plug the cerebral or femoral arteries, &c., causing hemiplegia, gangrene of foot, or other severe lesions, through simple obstruction. Ulceration may supervene on chronic plastic endocarditis and its symptoms be complicated with the obstructive effects of this; but it usually kills without dropsy. Sometimes in its typical form we have known it mistaken for continued fever.

Some say that pus may be found, as little abscesses, in the tissue of the valves under these circumstances, but of this we have no experience. We have, however, already given examples where pus in quantity was found in the heart's muscular walls when the process of ulceration extended from the endocardium to the tissue beneath.

Limitation of endocarditis to the valves and their neighbourhood.—We would repeat that all we have said as to acute endocarditis applies almost solely to the valves, and to such extensions from the valvular changes as arise through the spread of the ulcers to the attachments of the valve, or the friction of masses of fibrin on the neighbouring parts. Very occasionally we find a patch of nodular thickening of the endocardium in the left auricle, about the root of the mitral; and once or twice, under exceptional circumstances, as once over a partial rupture of the septum ventriculorum, we have seen inflammatory granulations form on the endocardium remote from a valve.

Chronic endocarditis.—We here again allude to those white fibrous patches about the interior of the left ventricle, generally near its apex, which we have already described as disease of the muscle of the heart, because in cutting into them you find they dip deeply into and even through the muscle and extend in it without reaching the inner surface at some parts; so that they evidently belong primarily to the muscle, not to the endocardium, which is smooth and depressed over them. We have mentioned the causation of aneurism of the heart by these patches.

Changes in the forms of the valves.—We must now consider the changes in the valves which affect their form and efficiency. These make up a set of conditions of vast importance quite independent of the causes which may have produced them. When the valves are seriously altered in shape they are liable to give rise to two distinct kinds of ill effect, first, in *obstructing* by their contraction or rigidity the opening they guard; and, second, in *allowing a reflux* of blood, which they are placed to prevent. In either of these ways they produce obstruction to the blood, or *ischæmia*, with its general consequences. The principal changes in the valves are as follows:

Spontaneous rupture of the valves.—The question has been much discussed whether the valves of the heart can be ruptured through violent action of the healthy heart. It cannot be denied that such a thing is possible, yet we think it occurs very rarely, if at all. As to evidence of such an accident, that would be very difficult to obtain, because the sufferer does not die at once from rupture of a valve, he lives some time and the valve inflames and the heart dilates and hypertrophies, so that what we find is a broken valve and an altered heart, and it is then left for us to infer which of these was the anterior in time.

Now, when we reflect on the mechanism of the heart as concerns its valves it will be seen that when it is in its natural form and healthy there would be no strain from the muscle tending to break its valves, for the strain on the aortic valves occurs only in the recoil of the aortic wall, which can hardly be supposed to be capable of splitting the valve; again, the strain on the mitral is relieved by the open aorta, into which the blood can freely flow. *But if the heart be at all dilated* then comes a strong strain on the mitral columns, which now are not of sufficient length to extend across the enlarged cavity of the heart. Under this strain they will be liable to snap through. This occurrence, namely, the snapping of an overstrained mitral tendon in a dilated heart, we believe to be a relatively very common cause of severe heart disease, converting the very bearable trouble of a moderate dilatation into a hopeless disablement. We have repeatedly traced this occurrence in the history of cases under our own observation. It happens usually either in young rheumatic subjects or in elderly people with dilated hearts due to gout or Bright's disease. The person has been subject to some palpitation and other signs of cardiac dilatation, or he has had rheumatic fever, but, perhaps, suddenly during exertion he is seized with severe cardiac symptoms, which continue until his death a few months afterwards. We then find some of the mitral cords severed, and sometimes very curiously twisted up into spiral knots surrounded by masses of vegetations, and, perhaps, causing ulceration of the endocardium by the pressure of these masses, which are very commonly found evidently dividing the neighbouring cords. Dr Albutt has drawn attention in an able paper in 'St George's Hospital Reports,' to which we would refer you, to some of the consequences of dilatation of the heart during exertion. We believe the division of the mitral cords may result from this simple cause. Such cases are of frequent occurrence, and they often receive another explanation, which we, however, cannot concur in. It is said that the laceration of the mitral occurs in the healthy heart, and that the left ventricle then dilates in consequence of regurgitation through the mitral orifice; but we have already stated our reasons for believing that regurgitation through the mitral does not cause dilated hypertrophy of the left ventricle, although it often accompanies it; the dilatation cannot be the effect of a presumed division of the cords in a healthy heart; it can only, we think, be explained as we have just explained it. However you may think the disease arises, the class of cases where mitral cords are divided and the left ventricle is dilated forms one of the most typical groups of the cases known, clinically, as "*regurgitant mitral,*" and distinguished from "*obstructed mitral,*" which latter cases always have another kind of origin, as we shall presently see.

Retroversion of the valves.—Again, you know that the valves are not made to meet at their edges, but their borders are turned up and meet in such a way that they mutually support each other in the action of the valve. But we often find that the borders of the valves have been bent back by the blood stream which they are placed to resist, so that the current they should check pushes their edges along with it, and escapes through between them. This condition is of less special importance in the mitral valve, where it is a mere accidental complication of more severe disorder already disabling the valve; for the structure of the valve, with its numerous cords holding every part of the surface below, prevents such a retroversion unless the cords are divided by disease. But in the case of the aortic valves we have often found such a bending back of the valve to have suddenly made a very strong man into a hopeless invalid; in this way—the strain of the heart somewhat widens the aortic orifice, so that the valve segments no longer sufficiently meet by their borders and support each other. This widening of the orifice is due to an irritative softening of the aortic coats, such as induces aneurism elsewhere. The valves not supporting each other in some unusual exertion, one of them bends down into the ventricle, and regurgitation is thus allowed. The same result may happen through inflammatory contraction and shortening of the valves, so that they will not well meet; regurgitation being free, and no obstruction existing, these form the very worst class of cases. They compose a considerable proportion of all cases of aortic regurgitation, and these cases usually occur in strong young male subjects, whose occupation is laborious, though it is occasionally met with in women. The history of sudden suffering during exertion in such subjects will often enable you to identify the cause during life. Sometimes the case is not so simple, for the failure of the valves to meet may be due to an actual aneurism forming about the root of a segment, either above or below its attachment.

Retroversion of an aortic valve, as well as rupture of the mitral cords, may occur as a consequence of endocardial ulceration. But the chief interest of these states lies in their occurrence as independent and recognisable original causes of grave heart disease. When they occur in the course of ulceration, however, they seriously add to the peril and suffering.

The occurrence of such ruptures and retroversions affords another instance, showing that the muscular force of the heart is the main cause of injury to its structures.

Deformity of the valves from ulceration.—We have described the effects of ulceration on the structure of the valves. Its effect on their figure and function is generally very destructive of the normal state of both. During the activity of the ulceration there are always present large masses of "vegetation" attached about the inflamed spots, and these obstruct the opening protected by the valve, while they also prevent its efficient closure, so that in ulcerative endocarditis both obstruction and regurgitation may be present at the affected orifice. But the obstruction, if there be no antecedent chronic mischief, does not reach such a pitch as to cause any considerable dropsy, while those other grave results of the ulcer which we have already described will often destroy life before there has been much time for the slower production of contraction, &c., that would follow if the ulceration were healed. Obstruction is generally most marked in these cases at the aortic orifice, which is more easily closed by a mass of vegetations than the mitral. Regurgitation is, on the other hand, often very considerable in these cases, especially through the mitral when its cords are divided. Ulcerative endocarditis may heal, leaving the valves much reduced by the destruction caused. Thus, we have seen a case where a large part of two aortic segments had been removed, and one where the aortic valves were reduced to relics.

Deformity from contraction, "ossification" (petrifaction), &c.—Contraction of the valves is generally the effect of that prolonged irritation which is the natural sequel of their acute inflammation; it is by far more common in the mitral valve than in the aortic. But the aortic valves do not escape in all cases; thus, we see the obstructive effects of inflammation of the aortic valves in two of them becoming adherent, although their efficiency may remain. If this union be perfect, the partition between them is in time dissolved, and the two valves become one, or an adhesion may take place between the edge of one and the aorta, and thus one valve becomes two. But in the mitral valve, owing to endocarditis, a thickening of all the parts included in the structure frequency occurs. Thus the curtains become three or four times their natural thickness, and form hard masses like cartilage; indeed, we have microscopic sections from specimens showing well-marked cartilage in such a thickened mitral; the orifice is altered in shape, becomes round, as you see here, and will scarcely admit the point of the finger; or more commonly it has the form of a button hole and may be no larger than a shirt-button hole; at the same time the tendinous cords become drawn up, shortened, and thickened, and sometimes several unite together into a thick tendinous mass. We have met with examples where all the cords had gathered into two short pillars of white fibre, one at each end of the mitral opening, which was half an inch long and one sixth of an inch broad, the whole structure looking like a little funnel composed of tendinous tissue. The endocardium on the septum of the ventricles may be much thickened close to the valves, and sometimes we meet with a very remarkable thick band, projecting into the cavity, as is seen here, as though it helped to maintain the column of blood, and so compensate for insufficiency in the valves; sometimes there are several smaller pieces, which may curiously resemble valves in their appearance.

In very old diseased valves *calcareous changes* may take place. A very favourite seat is the inner side of the mitral valve, between it and the aortic; but sometimes the whole mitral orifice is surrounded by a bony ring. In the aortic the deposit generally begins at the bottom of the valves, at their place of attachment; they are often large, so that at the post-mortem examination the finger will feel their presence at a time when they are not yet visible.

This petrifaction is the most frequent cause of simple aortic obstruction. The earthy concretions grow into great masses that invade the valves until at last they are converted into immovable nodular masses of stone, with the orifice of the aorta reduced to a little chink of the size and shape of the mouth of the uterus. Such examples you may sometimes meet as causes of the sudden death of hale-looking old men who never had dropsy, and sometimes they are found in the bodies of old men who have died of independent diseases, even when the obstruction of the aorta is nearly absolute; such cases are very surprising, and prove how comparatively innocent is simple obstruction of the orifices.

Fenestration of the valves.—There are still other changes found in the valves. We will first allude to the so-called fenestration of the valves. We refer, especially, to what you see here in these aortic and pulmonary sigmoids, and which you may every day meet with; that is, a perforated or fenestrated condition of them, above the crescentic line where they are apposed. The perforation can, therefore, produce little ill effect so long as the free border keeps its firmness and continuity so as to do its share in resisting tension; these small holes have often been looked upon as a result of atrophy, but there is little proof of this; we have seen them in young people, and we have always regarded them as congenital, for in some of the lower animals the sigmoid valves are attached to the artery by thin tendinous cords (which are produced here by the perforated condition), in the same way as the auriculo-ventricular. You may see this in these hearts of the shark.

True aneurism of the valves is sometimes met with. Thus, in the mitral valve, pouches may be seen opening towards the ventricular side: these are sacculated. There is also an aneurismal dilatation of the whole valve, as you see in this mitral, where both curtains bulge out into the ventricle, and as you may also see in this aortic valve, which is very largely distended.

Malformations of the valves.—We mention these last, for as, no doubt, they arise from inflammatory changes and adhesions, you will be prepared to know how they are produced. Thus, this funnel-shaped membrane, taking the place of the pulmonary valves, is evidently formed by their union, for the lines of junction can still be seen. This heart came from a girl who died in the hospital only a short time ago, and who never had any illness during her lifetime, and, therefore, there was every probability that the disease occurred in fœtal life; such cases of conical pulmonary valve are not very rare, they may reach elderly life. With this conical membrane at the pulmonary orifice, you may contrast the condition of the aortic valves in the same heart, and see what has occurred there from inflammatory adhesion; instead of all three becoming united, two only have so done, and in course of time the partition has been dissipated and the two have become one, and thus we account for a *deficiency of valves.* There can be no doubt whatever that this is the way in which these so-called malformations come about, for we think the same thing may occur in *adult* life as a result of inflammatory adhesion. We have here four hearts: in one you see two valves united as far as their middle, but yet the two free edges can be seen; in this one you see a perfect blending of the adherent sides, and the united structure has sunk down below the level of the valves; in this third specimen you might imagine you were still looking at the same at a further stage, for it exactly resembles it, only the uniting partition is much less, and scarcely divides the large valve into two; while in this fourth heart you see a large valve with only a trace of partition at its bottom, and thus two valves have been formed out of three. Here is a pulmonary artery with two, and here a good specimen of aorta with only two valves. *Supernumerary valves* are often formed in like manner. Thus, owing to an inflammatory adhesion, the free edge of the valve becomes fixed to the aorta, and thus two valves made out of one, producing four in all. This supernumerary one is much smaller than the others. Where the valves present quite a healthy appearance, and are uniform in size, it might be a question whether the excess arises in the way mentioned; but since four valves are much more frequent in the pulmonary artery than aorta, and as the former appears more susceptible of disease in uterine life than the latter, the regularity of the valves may be owing merely to the circumstance of the early period at which the adhesion takes place, *i.e.* when all the parts are plastic. Such perfect pulmonary and aortic valves you may see in these specimens. In the heart of the girl we have just shown you, with deformed aortic and pulmonary valves, you will see also that the mitral is malformed: this is rare.

We have twice seen the front segment of the mitral connected to the septum of the heart near the apex by a long, worm-like, fleshy band.

Valvular Obstruction and Regurgitation.—We believe that aortic obstruction without regurgitation is a rare form of heart disease, and that those authors who make it the most common are in error. Both aortic and mitral obstructions are generally the results of chronic sequelæ of inflammation or of calcareous degeneration, which may or may not be the consequence of inflammation. The distinction between the effects of regurgitation and obstruction is important; regurgitant disease is more formidable than obstructive. It is true that in a large proportion of cases both regurgitation and obstruction occur, but the effect on the heart will differ much according as the obstruction or the regurgitation prevails in the case. This may be stated as the rule—that the *cavity of the heart behind the affected valve hypertrophies in proportion to the obstruction and dilates in proportion to the regurgitation at that valve,* a very important difference, since the danger and suffering are in proportion to the dilatation only. We may say, then, that obstruction with its attendant hypertrophy is less formidable, while regurgitation with its attendant dilatation is more formidable. As to the relative danger of mitral and aortic disease generally, so much depends upon whether the disease is obstructive or regurgitant, and whether it is combined with ulceration, that we do not think any useful rule can be given. But as the aortic valve is more subject to retroversion, which allows a large unobstructed regurgitation, and therefore a great dilatation, the immediate dangers of aortic disease are greater. As a matter of experience we should say that *aortic valvular disease brings with it ten times more danger of sudden death than mitral disease,* and this is due to the ready retroversion of the valvular segments.

"Polypi"—Thrombosis or Ante-mortem Coagula in the Heart.—The comparison of the healthy heart with an aneurismal swelling appears always far fetched. When one is told that "like an aneurism, the heart is a dilatation in the course of a great blood-vessel" the statement seems a little absurd. Yet dilatation of the heart was on this principle formerly called aneurism or "general aneurism" of the heart, although that term is not now used. A relic of it is, however, preserved in those authors who apply to saccular aneurisms of the heart the term "partial aneurism" of the heart, for this name is given in opposition to "general aneurism," which is dilatation. There are good reasons why we should refuse the term aneurism to the dilated heart, because its dangers are not like those of aneurisms, namely, pressure and bursting, but the peculiar set of conditions due to cardiac obstruction. But, nevertheless, we should notice one point in which the dilated heart behaves like an aneurism, and that is the occurrence of coagulation of the blood within it; the coagulation is from the same cause in both instances, viz. stagnation dependent on the small proportion which the orifice bears to the size of the dilatation. We have already drawn your attention to the deposition of fibrin on the inflamed endocardium, making the great mass of "vegetations" in endocarditis. At that time we spoke of the conditions that favour such deposition of fibrin. The chief of these, however, are two—an uneven surface and stagnation of the current. To understand the common appearance of *ante-mortem* clots in the heart you must recall the appearance within its apex, &c., and remember the many recesses between the *columnæ carneæ;* many of these recesses form shut sacs; the existence of these would easily appear to be a constantly threatening source of danger from coagulation, but that in the systole of the natural heart they are closed completely and all the blood expelled from them. When, however, the heart is much dilated, these recesses in its wall do not entirely close in the systole, but remain as permanently patulous cavities, in which blood, if the general current is very slow, may be entirely motionless, and so be ready to coagulate. In this way it is that clots form in the recesses of the *columnæ carneæ* near the apex or appendix of the dilated cavity of the heart, especially the left ventricle and the right auricle. Once formed, the surface of the clot receives additions of fibrin layer by layer as any other unnatural surface in the blood stream always does, so that at last there is a considerable mass with a laminated structure outside, but containing some blood in its interior. You must distinguish the first coagulation of the *entire blood* from the deposition of *colourless fibrin* upon it. This process takes place in several neighbouring recesses at the same time, and some of the masses, as they grow out of their recesses, meet together and coalesce and thus are formed greater or smaller laminated bodies. The laminæ do not cover the whole surface concentrically, but each is only of partial extent, so that their edges show as sinuous lines upon the "polypus," as such a concretion is called when it projects into a cavity of the heart. It gets this name, "polypus," because of its resemblance to polypoid growths in mucous surfaces, being always rounded pretty smoothly on its free surface where subjected to the current, though it has root-like projections extending into the recesses it first formed in.

But all polypi are not so small in their beginnings. Some form by a more extensive coagulation of the blood in the apex or appendix, the clot so formed implicating the blood in the recesses around, so as to be held in its place by its ramifications among them. Over this clot layers of fibrin form as over the smaller ones, and then the look of those that arose very slowly and those that come more suddenly is very much alike. But the difference is in their interior. For all the while these polypi exist they are undergoing degenerative changes, which result in the softening down of their central parts to a pulp of creamy consistence. Now, in the slowly formed concretions, composed chiefly of fibrin, the creamy matter is white, but in the more hastily formed larger blood-clots the cream is coloured with the more or less decaying blood pigment, so as to be purple or brownish-purple. By the amount of blood pigment in the interior of these clots you may generally judge the size of the blood-clot which they were originally founded upon, although, no doubt, some amount of decolorisation of the clot is always going on, the red corpuscles breaking up, and the *liquor sanguinis* around washing out the pigment they leave. It is true that the small polypi contain least pigment, as if most thoroughly washed, but these are they that grow very slowly from deposit of colourless fibrin.

The action of the blood on the original large clot is much impeded by the layers of fibrin that surround it, so that the process of decolorisation proceeds but slowly. If you examine the contents of the softened polypus microscopically you find it chiefly composed of detritus of fibrin in an amorphous state, but it is not right to say that the microscope always proves that these concretions are not purulent, for sometimes we have found a great number of pus-like corpuscles in them which we think, perhaps, are due to the wandering of white corpuscles from the blood through the fibrin layer into the interior of the polypus. These polypi are not without their special dangers. They may be dislodged and pass on to obstruct the large arteries. Note, then, that in any case where you suspect embolism it is not enough to examine for a cardiac murmur, because these polypi may be found in dilated hearts where the valves are perfect, especially if there is Bright's disease, as we have often seen. Or a polypus may burst and throw its contents into the blood stream, leading to embolism. We have never seen decided examples of this, but cases are recorded. It is said by some that the result of this occurrence would be suppuration, &c., in the part embolised; we should not, however, expect this, since passively formed fibrin is inert in the tissue it reaches, and it is only when the fibrin comes from a seat of acute inflammation that it sets up inflammation.

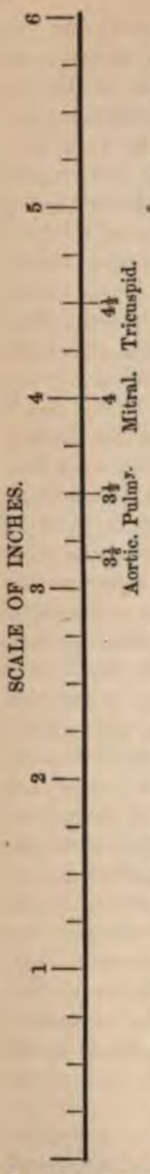
A hollow, collapsed, fibrin concretion is very like a polyp or sea anemone. It is a very strange-looking pathological object, although its explanation is so simple. Before Goodsir pointed out the passive nature of the process forming it, its pus-like contents naturally led to the idea that it was a purulent sac in the heart, and therefore very horrible and interesting; but the almost constant connection of it with cardiac dilatation rather than with suppurative disease might have suggested its true powers. However, it must not be assumed that it is always devoid of active powers. We have on two occasions found these polypi in the heart in pyæmia, and have several times met with suppurative thrombus (*i. e.* pus in the veins) in the places where great venous stagnation occurs, as in the pelvic plexus, &c. Again, the polypi do often, as we have said, contain a great many pus-like corpuscles, so that we do not think their origin can be held to be always necessarily passive. Remember, too, that cancer may spread in them. Rindfleisch speaks of their outer layers being better nourished than the inner, which assumes some activity in them, although he does not believe in the spread of cancer in them and takes their nature to be passive. We think there is evidence that such polypi may have active as well as passive causes—in other words, that the state of the blood itself is part cause of their formation. The question still awaits further elucidation.

We have spoken of two stages of slowness in the formation of antemortem clots in the heart, but there is a *third*, which is of more importance than either. This kind is not yet recognised, though we have drawn attention to it before. This is the process. A coagulum forms in the apex of the heart, and never has sufficient hold to keep its place, so that it is almost immediately dislodged and cast, fresh as it is, into the pulmonary artery from the right heart or into the aortic system from the left. It would be a great wonder if such detachment of loose clot did not occur. Such clots, however, when found in the vessels, are called " thrombosis " of the vessel [thrombus is clot formed *in sitû*, embolus is clot cast into the part from elsewhere], because no concretions, &c., are found in the heart to explain its presence. Such we believe to be the nature of the case given by Dr Dickinson as thrombosis of cerebral arteries, such also are the cases called puerperal thrombosis by several obstetric writers. They are clots formed in the heart and cast on loose into the circulation. We shall speak of this again under Arteries.

The blood in the heart after death.—You should remember that the right side of the heart, and the vessels leading from and thereto, contain naturally, after death, a coagulum such as would form in a basin during venesection. Thus, as the body lies on its back, the red corpuscles sink down and the fibrinous clot forms above them, and thus in right auricle and ventricle it is found, white or buff anteriorly, and red posteriorly ; and connected with these clots in the auricle are those proceeding from the vena cava and jugular vein, and from the pulmonary artery with its branches ; the shape of the valves being modelled upon the latter. On the left side the blood is less firmly coagulated, the auricle being filled with a dark semifluid blood, and the ventricle with a small quantity of the same, any coagulum found within it being very small. This is on account of the usually contracted state of the left ventricle. Also, it is more rare to be able to detach branching coagula from pulmonary veins. Why the blood should be dark and scarcely coagulated on the left side is not quite explained; but you must remember that the left heart lies low, so that it receives the corpuscles while the clear fibrin is in the arch of the aorta, as that lies on a higher level. In diseased heart with dilated left ventricle we may find a large softish clot, but seldom so well formed as on the right side. If we discover anything unusual in these clots, we shall more rightly regard them as the *consequence* of a peculiar mode of death, than as the cause of it. If a clot forms before death, it can generally be told from the fact of its being firm, adherent to the walls, and having lost its colour, also, when opened, it is softened in the centre; whereas we always find some evidence of the gravitation of the red corpuscles in clots formed after death, so that they are pale on the upper side and dark on the lower. A little experience will enable you to distinguish them; by the same means you will be able to distinguish an old clot from a recent one in a small vessel, as the cerebral to which we have already alluded; but it is not always easy to say whether a fibrinous concretion has been formed in a vessel, or whether it has been carried from a distant part. If it be supposed to have been brought from the heart, it must be compared with the vegetations there found. Its shape, and the mode in which it is placed in the vessel, will often show whether it has been formed there or not; for if coagulation occur from a disease in the tissue, all the vessels leading to it, up to a certain sized branch, will be found filled; whereas, if the fibrinous plug be carried from a distant part, it will lodge at that spot where its progress is arrested, and the smaller vessel beyond will of necessity be found empty. Under the heads Kidney and Spleen, we shall have occasion to speak of fibrinous masses found in them in connection with heart disease, and which are due to some portions of vegetations carried from the valves; they are more frequently found in connection with such vegetations than not, but very often in cardiac disease without such vegetations, and, therefore, the theory in this case is not without doubt in some minds.

A very interesting class of cases connected with this subject was mentioned by Simpson, in which during the parturient state there is a disposition for clots to appear in the femoral arteries, so that gangrene of an extremity sometimes takes place, or in the pulmonary artery, causing sudden death. We believe this class belongs to the sudden thrombosis of the ventricles, and embolism, which we have just described (p. 140).

This table is one which hangs in the post-mortem room, and which we have exchanged for the old one of Bouillaud. The measurements are taken from a great many examples; and we have given them in inches instead of in French lines, as by Peacock and others; they are in consequence, perhaps, not quite so accurate, but you will be better enabled to carry them in your memory.



MEASUREMENTS OF HEART.

Circumference at base	9 in. or rather more
Length	rather more than breadth
Circumference of R. ventricle . .	5 in., nearly
Length of right ventricle . . .	3¾ in., about
Thickness of walls of L. ventricle	½ in., about
„ „ R. ventricle	1-6th in., about
„ septum ventriculorum	intermediate between two above
„ left auricle	⅛ in., about
„ right auricle . . .	rather less than left
Orifice: L. auric.-ventric. (mitral)	4 in. in circumference
„ R. „ (tricuspid)	4½ in., or rather more
„ Aortic	3 1-6th in.
„ Pulmonary	3½ in.

It will be seen that the tricuspid orifice is larger than the mitral, the mitral than the pulmonary, and the pulmonary than the aortic. The pulmonary larger than the aortic by ⅛ part, and the mitral larger than the aortic almost by ¼ part (being ¾ inch larger) and the tricuspid is half as large again as the aortic.

These measurements were made by this graduated cone, which being inserted into the orifice, the line at which it is stopped is read off as the size of the opening; when there is much rigidity, and the valves have lost their pliancy, the instrument is useless; you must then lay open the orifice and measure across by a graduated tape, taking care to fit it into all the inequalities.

As regards the capacity of the cavities, each probably contains about three ounces of blood; but it is by no means certain, as some maintain, that the right is more capacious than the left; it is said, too, that the ventricles hold more than the auricles. The weight of the heart is about 9½ ounces; this is the average taken from all our cases during three years; as these are mostly cases of chronic disease, and the manifestly enlarged hearts are excluded, it is probable that in healthy persons the weight may be somewhat higher than this. In wasting diseases it weighs sometimes as low as four or five ounces; or in hypertrophy may reach to 2½ pounds; in the latter cases, the wall of the left ventricle may measure an inch or more in thickness, and the septum participates in the changes of the ventricles.

DISEASES OF THE ARTERIES

Malformation.—Irregularities of distribution concern principally the anatomist, and are treated of in anatomical works. We have already mentioned the most important malformations of the great vessels when describing those of the heart, to which they properly belong; we would, however, remind you of the congenital contraction of the aorta, and its constant position at the entrance of Botalli's duct.

Hypertrophy.—The arteries are very capable of enlargement, as we see in the increased size of collateral vessels after ligature of a main trunk, or in the gravid uterus; you will find, though, that in such cases the arterial walls are thin and vein-like. Thus, the enormous cervical and humeral arteries communicating between the arch of the aorta and its thoracic portion, in a case of congenital contraction of the aorta at the junction of the Botalli's duct, were much more like veins in the structure of their walls. The arteries are thick in elderly people, especially when the subjects of granular disease of the kidneys. This thickness is chiefly due to hypertrophy, but the coats are usually in a degenerated condition. There has been much rather keen discussion about this state of the arteries lately, Dr Geo. Johnson maintaining that the arteries of the body, in cases of granular kidney, have their proper coats greatly hypertrophied, while Sir W. Gull and Dr Sutton assert that they are thickened by a fibroid change in the outer coat especially, which these authors term "arterio-capillary fibrosis." We have found that the thickening of the vessel's wall is not so great nor so constant as was supposed in this discussion, but any considerable thickening is accompanied by degeneration of the coats, and very rarely is a simple hypertrophy.

There is, however, great hypertrophy of the *renal* arteries in granular disease of the kidney, just as there is hypertrophy of the hepatic arteries in cirrhosis of the liver, and of the bronchial arteries in old fibrous disease of the lung.

Atrophy.—It is said by Virchow that in chlorosis the whole vascular system, including the arteries, is in an atrophied condition similar to that which prevails in the blood. This condition has been thought to be congenital in chlorotic persons. We have no special experience on this subject, but the point raised is one of great interest.

The aorta may be very small in cases of obstructive disease preventing free access of blood to the left heart, especially in congenital contraction of pulmonary orifices.

Inflammation—Arteritis.—The remarks we made upon the general and comparative nature of endocarditis (p. 127) apply to inflammations of the arteries and veins, since their lining membrane is likewise devoid of vessels. It is true of the arteries as it is true of the heart, that we do not meet with a general acute inflammation of the interior, such as would resemble a general pericarditis or pleurisy in producing lymph over large tracts of surface.

Experiments on blood-vessels have failed in developing those well-marked phenomena of inflammation which are seen in ordinary serous membranes, and thus, in those cases where a vessel is found filled with fibrin, adherent to the walls, it is probable that much of this is due to a mere coagulation of the blood, and not to an exudation from the arterial surface.

Acute general arteritis.—It was formerly thought that the deep red staining, which we now know to be due to imbibition of hæmatin through solution of the corpuscles of the blood, was a sign of inflammation; but even when it had been clearly proved that such staining caused a redness, it was still maintained that some redness was of an inflammatory kind. But this belief is now entirely abandoned, and the accounts of acute aortitis given by Peter Frank and others are recognised as erroneous; they were founded on descriptions of the symptoms preceding death in cases where early solution of the corpuscles occurred. We may say that general acute arteritis is unknown.

Chronic general arteritis.—Cases are occasionally met with where the great arteries, in young and vigorous subjects, become obstructed, so that no pulsating vessel can be felt during life in any of the limbs, and the arteries are found thickened and containing adherent clots after death. In other cases the same condition may be less general, yet extensively distributed. The character of these cases is not agreed on; but we have found extreme examples of such disease in the arteries to coexist with the fibrous thickenings of myocarditis in the left ventricle, suggesting plainly that the arterial disease is of the same inflammatory nature, and that a general chronic inflammation of the interior of the vascular system was present. In some of these cases the disease has curiously selected corresponding arteries at their corresponding parts. Thus, both the femoral and both the brachial arteries may be thickened and stopped up with clots when no other arteries are implicated. Such a condition brings great danger of gangrene of the limbs in which it occurs. It is fortunately very rare, but must be borne in mind as occasionally met with.

For instance, a girl was lately in the hospital in whom no pulsating artery could be felt in any part of the body, and this state had been coming on for some years; after death, all the larger trunks were found to be narrowed or obliterated.

Circumscribed arteritis.—This may be *ulcerative.* We need only allude shortly to the attack of neighbouring ulceration on the arterial coats, though the clinical importance of this dangerous occurrence is very great. It is a frequent cause of death. Thus, for instance, a bubo, or an ulceration beneath the skin extending from a diseased knee-joint, may open the femoral artery, or severe ulceration about the fauces may reach the carotid, or an ulcer on the face open the facial artery. More frequently an ulcerous state of the stump after amputation may eat into the main artery of the limb. Another important and not infrequent example of such ulceration is furnished by the spread of stomach ulcers, so that, through adhesion of the stomach, they reach and open the splenic or some other neighbouring artery. When an ulcer reaches an artery the wall of this becomes thin and brown, and at length the vessel is burst by the pressure within. We have met with an example where, the splenic artery being thus attacked by gastric ulcer, a clot formed within it at the affected spot, but was pushed on as an embolus into the spleen. This occurrence might be regarded as an attempt, though unsuccessful, to close the vessel and avert the danger.

Another method of origin for ulceration of an artery is that which is sometimes found in the spread of ulcerative endocarditis; here we may see an erosion of the wall of the aorta, at a point struck by a hard, freely moving vegetation on a segment of the aortic valve. Sometimes patches covered with vegetation, and evidently acutely inflamed, but without loss of tissue, are found in the ascending aorta when its valves are affected with ulcerative or otherwise severe inflammation. The great danger of these occurrences lies in their tendency to displace the valve, and so retrovert one or more of its segments.

Next we must notice a degree of inflammation which we will call *inflammatory mollities.* In this there is no ulceration; it leads to softening and swelling of the arterial wall, and so may induce either rupture or aneurism. It is important to notice that such an inflammation may arise from embolism under a certain defined condition, to which enough attention has not been given. This condition is that the embolus must come from a seat of inflammation; such an inflammatory embolus, reaching into and lodging in a smaller artery, such as the femoral, meningeal, or any other, inflames and softens its coat, so that the vessel either bursts or yields to make an aneurism. Embolism of arteries is very frequent, but it does not often give rise to aneurism or effusion of blood, because the embolus is not inflammatory, except in rare cases of ulcerative endocarditis. The aorta is the most frequent seat of this inflammatory mollities. Here it is generally spontaneous, or, at least, arises in the aortic wall as a proper disease of its tissue.

Inflammatory mollities is the common cause of aneurism in young subjects, but it is not limited to early years. It may also induce rupture of the aorta, or, by being near the root of an aortic valve it may allow the valve to turn over. This is one of its greatest dangers in young hard-working men. The affected part of the vessel is generally sharply circumscribed, and when the vessel is opened it bulges inwards, because the swelling of the coat enlarges its extent of surface; prior to the opening of the vessel this part had bulged outwards, and was, in short, already an incipient aneurism. Such a patch no longer has its proper yellowish opacity, but it is of a bluish moist appearance, and between your fingers it feels flabby and inelastic, more like wet membrane than like aorta, so that one can trace it by the fingers with one's eyes shut. The microscope shows that an inflammatory action is present, there are exudation cells in more or less abundance throughout the wall of the vessel, often in all its coats, and we have seen such cells collected in the outer coat of such an artery into small aggregations like tubercles in appearance (see 'Guy's Hospital Reports,' 1871, 1873). Though we ourselves have never seen anything that we should call "pus" in these arteries, yet it appears this has been noticed by other observers, particularly Mr Lawson ('Army Medical Reports,' 1866), who observed that phlegmonous inflammation of limited portions of walls of the arteries or veins is by no means rare at the Cape, where it leads to aneurism or rupture of such vessels. This implication of all the coats, including the adventitia, explains why, as a sequel, the artery comes in these cases to be firmly fastened to parts around it. A contraction of the lymph, as in other inflamed parts, may seriously narrow the calibre of the aorta. We have seen cases of death from this contraction. It is interesting to observe the distribution of this inflammatory mollities in the aorta; it is scattered irregularly throughout in circumscribed patches, reminding irresistibly of a cutaneous eruption, so that it seems fair to regard the acute forms, at least, as eruptive in their nature, that is, as local actions dependent on a peculiar general state.

Next we will notice a lower kind of inflammation, a *semi-cartilaginous thickening*, chiefly limited to the inner coats. This shows gradations connecting it with the inflammatory mollities we have just described; it appears in the deep layer of the inner coat as a very slow swelling, gradually elevating the surface of the affected part, which at the same time grows tough and inelastic, acquiring very much the same bluish cartilage-like appearance we mentioned as present in mollities, so that the two conditions are apt to be confused together as semi-cartilaginous thickening. The microscopic appearance in this state differs from that in the softer and more acute form in showing more of a hyaline or slightly fibrillated matter in it, with much fewer cells—that is, it differs as slighter inflammations from severer. If you study the borders of the patch you will see that the hyaline material arises in a swelling of the fenestrated lamellæ of the *intima*, with a multiplication of its cells.

These patches are always of slow growth, and when seen they show all stages of development together. Of their decay into a fatty paste or "atheroma" we shall speak presently. They may acquire an indurated condition by slow organization if time be allowed them. Their substance is like that of the proper inner arterial coat, and although not perfectly like it, yet they offer in this stage a very beautiful example of the development of inflammatory products to the likeness of the part they were produced in.

Syphilitic arteritis.—Several observers, especially those connected with the army, have concluded that syphilis is a common cause of disease of the aorta. The descriptions given by them, however, do not serve to distinguish their cases from the forms of inflammation of the aorta above described, and we have not been successful in distinctly tracing these states to syphilis. It is remarkable that they do not occur in prostitutes or generally in syphilitic females.

But we have met with very characteristic examples of a gummous inflammation of smaller arteries, especially of those within the cranium. Attention has also been directed to these by Dr. Hughlings Jackson. The change appears as a circumscribed yellowish thickening of the affected vessel; several times it has been in the basilar artery, implicating chiefly its outer coats. It is apt to cause softening of the brain.

Degeneration of the Arterial Walls.—*Endarteritis deformans.*—These are generally the results of chronic arteritis. Thus, the *fibroid* or *semi-cartilaginous thickening*, as we have already intimated, is rather a state left by the slow arteritis that once caused it than a sign of action present at the period of death. When such processes as we have just described have lasted long in elderly people and spread very extensively throughout the aorta—their chief seat—then the semi-cartilaginous thickenings are found to have undergone retrograde changes, softening them to yellowish pulpy patches, or hardening them to stone; meanwhile, the artery is wide and irregular from partial yielding of weakened spots, so that its interior may be greatly defaced. Extreme examples of this are really quite startling when seen for the first time, and contrast curiously with the perhaps entire absence of any symptoms that would prepare you for such a scene. The aorta looks, if we may so speak, like the skin of a dirty old eczematous arm turned inside out, the artery widening until it reaches three and a half inches or more in diameter, and its great branches equalling the aorta itself in size, the whole condition well meriting the name *endarteritis deformans* used for it by Virchow.

The retrograde changes in inflamed arteries are of two kinds, called *atheroma* and *ossification*.

Atheroma commences in the semi-cartilaginous thickening as a fatty degeneration. We have already seen that this thickening affects the inner coat of the artery, so that it is here that the fatty change begins. The fat appears in the form of grains occupying the spindle-shaped spaces that contain the cells in the tissue; soon the fat quite hides the cells, and at last it accumulates so in their spaces that these break into each other, the intervening tissue wearing away meanwhile; so that at last a considerable soft fat patch is found covered inside by the remains of the internal coat, between which and the middle coat it lies. Chemical changes then occur in the fatty detritus, resulting in the production of a quantity of cholesterine, which gives a glistening look to the matter when it is brought to view, so that it appears as a grey, rather glittering paste, which under the microscope shows only fat-grains and fragments of texture and cells with crystals of cholesterine. The accumulation of these products causes the patch to swell beyond its original size, and at length it may burst into the aorta. The blood then enters and washes out its contents. After thus finding a way into the wall behind the inner coat, the blood may be forced along between the coats, producing a dissecting aneurism. If a fibrinous coagulum then forms, the inner coat being pushed inwards, a complete stoppage of the circulation may occur. In this specimen you will see the aorta so obstructed, causing gangrene of the extremities; and in this one there are two distinct obstructions, one above and the other below the diaphragm. In this one, also, you will see complete obstruction at the lower end of the aorta; the coats have separated, a fibrinous concretion has taken its place within and between them, and thus a stoppage of the circulation occurred at this spot. In some cases the vessel may rupture and blood break through, causing instant death; and this has also happened, though rarely, in a limb. It is not so common, however, as the blood running between the coats. It was the contemplation of such cases as we have shown you which first suggested to Sir A. Cooper the possibility of putting a ligature on the aorta.

Ossification.—But, fortunately, all atheromatous patches do not soften down and rupture in the way we have just described. In by far the greater number of the patches the fatty and granular paste soon undergoes a greater and greater addition of lime salts together with disappearance of the fat, until at length a firm plate of bone-like appearance and consistency is found, forming what is called ossification of the artery. It is doubtful whether the fatty degeneration which constitutes atheroma always affects the semi-cartilaginous patch before it is converted into a bony plate. We rather think that in many cases the change from semi-cartilaginous thickening to the bony plate is effected directly by the simple deposition of lime salts in the still firm, half-cartilaginous tissue.

In these hard plates or "*ossification*" of arteries of which we have been speaking no true bone exists; indeed, it is true that in all the cretifications connected with the heart and arteries (unlike those of the cerebral and spinal membranes, which we shall soon mention) no true bone is ever found. If you take one of these hard plates from the aorta, and grind it down so that it will form a fit object for the microscope, you will see no lacunæ or other constituents of bone, but merely a number of round masses, disposed in a matrix of fibrous tissue.

The roughness of the inside of the artery, caused by atheromatous and bony changes, may induce coagulation of blood upon it, so that considerable masses of clot may be found clinging or hanging to the surface; such masses of clot may be detached and pass on to obstruct smaller arteries, passing, say, from the aorta to the femoral artery. Such an occurrence is not very uncommon as a cause of senile gangrene.

The three conditions, semi-cartilaginous thickening, atheroma, and ossification, are, then, the successive effects of the low inflammation for which the name *endarteritis deformans* is used by German authors In our description of them we have hitherto kept in view their appearance as presented in the aorta. The middle-sized and smaller arteries show changes essentially similar, which, however, most usually do not go far in the atheromatous, or reach the ossifying stage. These changes are especially frequent in the arteries of the brain, spleen, and heart. In the brain they are the common causes of apoplexy and softening; occasionally a diseased coronary artery has been known to burst into the pericardium. The form usually met is a thickening of the internal coat, having all the characters of the semi-cartilaginous change before described. The thick coat is generally rendered opaque by some degree of fatty degeneration, but is not so fatty as to be softened; indeed, these arteries certainly often become calcareous without a preceding stage of fatty softening. The thick internal coat is usually very easily separated from the others, so that you may take from the inside of such a cerebral artery a tube like a quill, leaving the artery apparently improved by the process, but really now only consisting of the middle and external coat.

While thus comparing these states of the smaller and greater arteries we should draw your attention to the fact that the aorta may be exceedingly diseased when the cerebral and other arteries of corresponding ize are almost normal. We have placed in the museum some examples to prove this. It is likewise true that the cerebral or coronary arteries may be much diseased while the aorta is normal. The radial artery also may be sound when the cerebral arteries, &c., are bad, and the same is true *vice versâ*. We have before mentioned the occurrence of thickenings of the renal, hepatic, and other arteries along with diseases of the viscera they supply.

The vast importance, variety, and universality of the results of this disease make it necessary that we should have a clear view of its causes. It was long ago suggested that the true cause of "atheroma" is overstrain, and this view was held by Dittrich before the irritative earlier stage was known, and in this country the same was taught by Kirkes, and afterwards was advocated by Conway Evans. Careful consideration of our experience convinces us that this is the true cause.

The reasons which tend to induce this belief that mechanical strain is the main cause of atheroma of the arteries may, we think, be enumerated with advantage; many of these reasons, which we think amount in all to very sufficient proof, have been urged before, but we hope to show that some facts that are often thought to oppose this belief are really in its favour.

1. The male sex shows greater liability than the female, and men are accustomed to more laborious work. It is, we think, true also that when women are the subjects of atheroma they have been used to an unusual amount of such work.

2. Whether this be true as among females or not, it is certain that the portion of the male population who evidence the greatest amount of atheroma are those who use the greatest muscular exertion, *e. g.* sawyers, oarsmen, soldiers, persons accustomed to excessive athletic exercises, &c.

3. Those diseases that diminish the volume of blood, and the consequent pressure within the arteries, prevent almost entirely the occurrence of atheroma of the arteries, *e. g.* phthisis, mitral obstruction, &c.

4. The pulmonary system of vessels escapes the liability to atheroma almost entirely. This has been the ground for starting a theory that venous blood has some kind of opposing influence, or arterial blood has some kind of favouring influence, on the development of atheroma. But a fact is often met which counteracts this suggestion of an alibi, namely, that when the right heart is greatly hypertrophied from chronic bronchitis or mitral disease, the pulmonary artery then gets commonly affected with atheroma, although its blood is more venous than usual under these circumstances.

5. The earliest appearances of chronic atheroma in arteries occur at points where the strain upon the coats is greatest. The obvious example of this is in the case of the convexity of the aortic arch, which is very exceptionally liable, as is the bifurcation of the vessel. But

there are other examples of this truth which have escaped notice, *e. g.* the cerebral, splenic, and cardiac arteries.

The peculiar liability of these vessels to atheroma is generally admitted, and it is often supposed to prove that mechanical strain cannot be the determining cause of the atheroma. But if we consider the circulation of the brain, heart, and spleen, we shall find peculiarities suggesting very plainly that their circulation is carried on under especial mechanical tension. Thus, the *brain*, by means of its *superior cerebral veins*, discharges its blood forwards into the longitudinal sinus, in a direction opposed to the current in the sinus which runs backwards; this must make the escape from the brain of the blood sent into it to be difficult, and thus lead to resistance to the flow from the arteries and tension within them, straining them, spoiling their nutrition by irritation, and so leading to "atheroma," and at last choking or bursting them when the chronic impediment has lasted years enough. The measure of this tension within the cranial circulation we shall show to be expressed in the Pacchionian bodies, which are hypertrophies of tissue from congestion around the sites of exit of cerebral vessels; other arrangements in the mechanism of the cranial contents, such as are contrived to maintain the necessary constant fulness of the cranium, must aid in inducing resistance to the free current of blood in the brain. The *heart* is very peculiar in its parietal circulation, seeing that the ventricles' contraction throws blood into the *coronary arteries*, while it hardens and compresses the tissue of the heart, so as to impede at the same moment the passage of blood on through the capillaries, thus producing tension in the artery from the resisted current. This, we think, is sufficient to lead us to anticipate that the heart's vessels would, sooner than others, suffer from any weakness, such as atheroma, which pressure elicits. Indeed, when we reflect to discover the direct causation of the impediment to the arterial blood in the athlete, we must find it to be principally due to the opposition to the arterial flow in his muscles during their over-strong and over-long contraction. The heart, then, is all one's life suffering as an athlete. The *spleen's* circulation shows remarkable peculiarities, for great venous spaces are constructed in it, which in a sense are obliged to wait the pleasure of the liver that they may pass on their current. We know how easily the spleen swells under hepatic obstruction. Some believe that its office is to form a sort of reservoir of venous blood, taking, during quiescence of alimentation, that flow which then is not needed in the digestive viscera. Whatever extent of this kind of view be true, it is certain that the spleen has, in playing its part, to endure an impediment to the onflow of its venous blood, and this will surely entail a resistance and tension in the *splenic artery* reduced, of course, by the tolerably ready distensibility of the spaces in question, and of the whole organ with them. This tension will strain the artery and lead naturally, on the view we are advocating, to atheromatous disease of its vessels, to which it is very liable. The other day Mr R. J. Pye-Smith, while acting as post-mortem clerk, pointed out to us the occurrence of two small aneurisms on two of the divisions of the splenic artery, close to the spleen, the artery itself being very bad. Now, if we look around to find any other organs whose circulation suffers the like peculiar strain, we do not find that we discover any; so that we think it is peculiarly strong evidence of the belief we are wishing to support, that these three organs should show such great and unusual liability to arterial disease. The only other organ which shows (but under prescribed conditions) much of such excessive liability to atheroma of its arteries is the *kidney*. This organ, however, only shows the affection in chronic Bright's disease, with contraction, and wasting of its cortex. Now, this disease goes with wasting away of the capillary glomerules in the Malpighian corpuscles of the kidney. One example, in which we made the observation very carefully, showed, in a fine section of the cortex, thirty-two wasted and closed-up glomerules to eighteen healthy ones; now, this closure of glomerules must greatly increase the strain in the renal artery, and thus explains why, in old Bright's disease, the artery thickens and becomes atheromatous.

6. The arteries of the lower extremities are more liable than those of the upper to atheroma. Now, it may be thought that, as the lower limbs are more constantly directed downwards than the upper, the strain in their arteries would be alleviated by the gravitation of their current, but this notion disappears when we remember that the current in the veins has a longer distance to achieve, under difficulties to which those of the upper are strangers—difficulties partly from gravitation, and partly from the varying and often rather great pressure of the abdominal viscera and their contents on the cava, &c.; pressures that bring with them at last more or less varicosity of the veins, which is the correlative and evidence of strain within the artery, such as tends to widen and render similarly tortuous the arteries themselves. Under these circumstances the greater liability of the femoral arteries than the brachial to atheroma is much in favour of the belief that mechanical tension is the cause of the atheroma.

7. The appearance of the interior of the aorta in the early stages of atheroma supports the evidence that the occurrence of it is due to tension within the vessel, for the atheroma is generally not distributed without a certain suggestive pattern. It observes this rule, that the atheroma forms lines lengthwise to the vessel's course, that is, across its transverse girth. Now, the longitudinal tension in the vessel's wall is not so great as the transverse, if only for this reason, that the tension wave of the pulse passes as a transient moving strain down the vessel, so that the longitudinal strain pulls on the unstrained part which the strain has left or not yet reached, and so is relieved, while the trans-

verse strain is all round alike, and has no escapement. Now, this transverse strain will, of course, tend to tear the coats across its own direction, that is, it will tend to tear longitudinal rents; but though it does not effect this, it produces chronic longitudinal weaknesses and irritations that evince themselves as atheromatous patches.

We might carry much farther the argument, showing that those chronic blood impurities, the chemical diatheses, uric and oxalic especially, go with a tendency to thick and strained vessels, and so at last with atheroma. All this, probably, through the resistance of impure blood meets in the capillaries and the tension in the arteries which this resistance creates, so that dyspepsia thus becomes a source of atheroma, through the medium of mechanical strain.

It seems to us that evidence enough has been given—1. That what is called atheroma of arteries is sub-inflammation of various degrees, of which the lower degrees end in fatty degeneration of the coats, along with the inflammatory products; and 2. That the determining cause of the occurrence of this change is mechanical strain. This by no means interferes with any belief that a general altered nutrition, in gout, syphilis, &c., may lay the coats of vessels more open to suffer from the said strain, and we are disposed to think that it is probably true that they do so; but no one has yet shown this to be true.

Besides the fatty and calcareous degenerations of the arterial walls which we have just now shown you as the results of deforming arteritis, you meet with simple fatty and calcareous degenerations which are primary and not preceded by inflammation.

Simple fatty degeneration appears in the form of opaque whitish spots marbling the inner surface, which at such spots is scarcely elevated if at all. Microscopically the change is seen to consist of a deposit of fat in the cells of the innermost layers of the inner coat. These cells are of stellate figure, and their branches intercommunicate. When healthy they are not easy to see, but when thus loaded with fat they become very conspicuous, so that the inner layer of such a vessel, when stripped off, forms a striking microscopic object. The further progress of this change leads to giving way of the fatty tissue, so that there is a very shallow breach of the surface, which is slightly downy at the affected spot, but the substance of the wall of the vessel beneath is practically unaltered. On the whole this simple fatty degeneration is not an important disease; it is very common in the aorta; indeed, it is rarely that you meet an aorta of more than two or three years of age that does not show some of it. The change is not often seen, however, in the middle-sized arteries, especially those of the limbs. But in the smallest vessels distributed within some organs, especially the brain, a fatty degeneration, shown in the collection of fat-grains around the nuclei of the cells in the *adventitia*, is very common in later life. This is a more serious disease; it leads to rupture of the vessels, producing apoplexy,

&c. It is a disease of the outer coat. These changes belong, however, to the proper pathology of the organs in question.

Simple calcareous degeneration is another mode of senescence in arteries; it is found chiefly in the middle coats of those of medium size, especially in the lower limbs. It affects the muscular fibres, and hence appears as bands running partly around the vessel, which at the same time is irregularly dilated through loss of its elasticity; the bands may form complete rings, and if the change progress to an extreme extent the artery may be transformed into a stony tube. Then arises a danger that coagulation within the vessel will cause senile gangrene.

Thus, as to the simple degenerations, it appears that the internal coat of the largest vessels is more liable to simple fatty degeneration. The middle coat of the medium-sized vessels is more liable to calcareous degeneration, while the outer coat of the smallest vessels is more liable to fatty degeneration.

While speaking of degenerations of the vessels we must draw your attention to the curious liability of the smallest arteries of some organs, as the spleen, kidney, &c., (see Lardaceous spleen), to *lardaceous disease*, so that the arteries exhibit the change before it is seen in any of the other components of those organs, and remind you that this liability does not extend to the larger arteries. Indeed, the small arteries escape entirely in parts whose tissue is not liable to lardaceous change, so that the disease evidently belongs to the organ affected, and the implication of the arteries so early in its course is an interesting instance, showing how the mode of nutrition of an organ governs the nutrition of the apparently independent tissues that enter to supply it. It is only the main arteries that have completely independent laws of maintenance, like the Roman *primæ viæ*. The small arteries, like the *viæ vicinales*, are under the control of the neighbourhoods they serve. Their diseases are commonly due to special faults of the organs they supply, while the aorta and its great branches have their own independent causes of disorder.

Aneurism.—Although you all, no doubt, understand what is meant by an aneurism (a sac communicating with an artery), yet the term is somewhat difficult to define accurately; and this is not so much on account of any obscurity in the disease itself, as because authors have adopted such various names in connection with it, and sometimes have used the same terms in opposite senses. This is especially the case with reference to the terms *true* and *false*, which so puzzle students, owing to their different and even opposite applications to aneurism. There are also various names given to aneurisms, some denoting the arrangement of the arterial coats, others the form and size of the sac. First, with respect to the terms *true* and *false*, and the manner in which they have been used. Scarpa, one of the earliest exact writers on the subject, from failing to find all the coats of the vessel in those aneurismal pouches which form tumours and contain *ante-mortem* coagula, described this absence of the coats as the true condition of aneurisms; whereas he called that a dilatation, or false aneurism, where all the coats remained, and no coagulation had occurred. Now, it is remarkable that the very opposite terms have been given by many authors to the same conditions, and aneurisms have been called true where all the coats are perfect, and false where some of the coats were wanting, such authors evidently being influenced by the connection in their own minds of true with perfect and false with imperfect. But the idea of Scarpa has a certain practical value, at least so far as it applies to aneurisms of the aorta, for no two affections can be more different than the dilatation of the whole vessel on the one hand and the formation of a pouch from its side on the other. The one case interferes with the circulation and the heart's action, while the other only produces effects of pressure or mechanical thrust on surrounding parts, like an ordinary tumour; the one consists, as Scarpa truly said, of all the coats, and contains no coagula, while the other, if of any size, is deficient in the coats, and contains coagula. Although he was so far right, it certainly was incorrect to limit the name true aneurism to that only where the coats were deficient, for the early stage of an aneurism is generally a bulging of the whole arterial wall at the spot affected (the exceptions being rare instances of rupture of the artery). So that, at first, all the coats of the vessel may be detected in the wall of the sac, though truly in a very altered condition, and very soon disappearing, so that you cannot find the middle coat in a sac bigger than a plum. The forms or shapes of aneurisms give rise to many names for them. Thus, when the whole circumference of the artery is dilated, and the enlargement circumscribed, the term *fusiform*, *cylindrical*, or *globular*, is used. Sometimes such a dilatation of a limited part of the whole trunk is abrupt, reaching suddenly a large diameter; it then may turn over the border of the opening in the parent vessel, and extend up or down it a greater or shorter distance, so that the opening of the vessel makes a projecting lip within the sac. The aneurism is then said to be *invaginated*. The term *saccular* is applied to the commoner form of aneurism, where a pouch comes off from one side of the vessel; but instead of using the invidious and contested term *true*, we think it would be better to employ the word *entire* to express the condition in which the entire wall of the vessel is bulged out. We then can use the ordinary word *false* for aneurisms in which the coats are not expanded, but burst through, that is, where none of the artery is in the sac it apparently gives off. We can also continue to employ the classical term *mixed* for cases in which an entire aneurism has burst at some point, so that a false sac forms beyond it.

Finally, when upon rupture of the sac the blood is dispersed among the tissues without a containing boundary—a very dangerous state—the old word *diffused* cannot be improved on to signify the accident.

Sometimes you see saccular aneurisms come off from fusiform ones, &c. Indeed, endless combinations of all these varieties may be met with in the aorta, when the arterial disease which has given rise to all these alterations of form is extensive and severe. Practically, if the sac of the aneurism be of some size, it is formed principally by the external cellular coat, the internal coat proceeding only a short distance inwards; within this you find fibrinous laminæ, which form in the part of the sac farthest from its mouth, as concentric layers. These laminæ are of a pinkish colour, are very thin, and lie one upon another in great number, so that a section resembles that of an onion. Their surface presents a remarkable appearance, from there being a series of parallel ridges upon them, resembling the "ribbed sea-sand." Their substance is not unlike that of the internal arterial coat, but they are apt to soften down like other old fibrinous clots, and sometimes such clot will move on out of the aneurism and plug the artery beyond.

The amount of this laminated deposit increases as the blood within the sac is allowed to rest, so that in fusiform aneurisms, where the blood freely courses throughout there is none of it. But in saccular aneurisms there is a varying quantity, the amount being large when the sac has a mouth that is small in proportion to its space; when the mouth of the sac is large and the sac small, so that the blood rushes freely in and out through it, then there is very little clot formed. The operation of ligature or pressure aims at securing an entire cessation of motion in the sac. It is said that sometimes the aneurism bends over on to the proximal side of the artery so as to compress it and thus shut off the access of the current, and cure itself. But against the chance of this we must reflect that the very pressure closing the artery would be the same pressure as that within the artery it is supposed to close, and, in fact, instances of this occurrence are as difficult to find as this reflection would suggest.

As aneurisms of the aorta are the most important, we will show you some of the best-marked specimens. In this one you will see a small pouch between the aortic valves, and projecting upwards between the auricle and ventricle; such aneurisms we think are produced by endocarditis where the valves are involved, and are similar to those we have already described under the term acute aneurism of the heart, for we can scarcely call a pouch between and under the valves an aneurism of the aorta. The first aneurisms which we can say occur in the aorta are immediately above the valves, in the sinuses of Valsalva; these are sacculated, have small openings, and generally give way before they have reached any great dimensions. Thus you see in this heart such an aneurism opening into the right ventricle just below the sigmoid valves, the patient's life was here protracted for a considerable time. In this you will see an aneurism protruding into the right ventricle and pulmonary artery, and sometimes such an one bursts into the artery, or such aneurisms may burst into the left auricle or the right auricle or right ventricle. More usually these pouches at the beginning of the ascending aorta rupture into the pericardium, as you see here, and instant death is the consequence. On opening the pericardium in such cases, you first of all see only the serum, which has separated; but on removing this the heart is found surrounded by a black coagulum of blood, which has come through a hole at the root of the aorta. The loss of blood is not much, and, therefore, the body is not blanched, as is so often the case from ruptured aneurism elsewhere; for, in this case, death arises from mechanical interference with the heart's action, and the blood is prevented flowing from distant parts, and therefore you meet the apparent anomaly, that death has been caused by a ruptured blood-vessel, and yet the tissues are found congested. In like manner distinct pouches form higher up, until we reach the lower part of the arch, where the vessel meets the spine, a very favorite seat for the disease. Aneurisms take different directions according as they are situated in the ascending or descending part, and, consequently, press upon or rupture into various parts, as the œsophagus, or the trachea or bronchi. One very important effect produced by aneurisms in this part is due to the pressure they exert on the pneumogastric and recurrent laryngeal nerves, and in the latter case especially, paralysis of the muscles of the larynx takes place, whereby stridulous breathing and other characteristic laryngeal symptoms are produced. Thus you will see in this specimen how the nerve was compressed; the symptoms were wholly laryngeal, and suggested tracheotomy, though it was not performed, as the nature of the case was suspected. In this other specimen, the operation took place. In some instances, where a large aneurismal pouch forms in the ascending aorta, it bulges forward and makes its way through the ribs and sternum. This is the least painful form, for it is often unattended by any pressure on important neighbouring organs; and in the very last case you saw in the post-mortem room the aneurism was so large that it not only penetrated the walls of the chest in front, but eroded the spine behind. General dilatation of the arch, or fusiform aneurism, you may see in these specimens. After leaving the arch, a very favorite seat for aneurism is where the vessel meets the spine, and is in contact with the fourth, fifth, or sixth dorsal vertebra. The aneurism may form on the front, side, or back of the vessel; though when large, very much the same effects are produced, as the enlargement takes place backwards and laterally. One of the most marked effects seen as the result of aneurism at this spot is a corroding of the dorsal vertebræ, so that when the vessel is removed their bodies may be seen quite eaten away, leaving the cartilages projecting and unaffected. In some cases the destruction of the bones may go on to the extent of laying open the spinal canal, so that, as in this case, the finger could be passed from the aneurism into the canal, and the patient, as you may suppose, had paraplegic symptoms. In such cases the wall of the aneurism has long disappeared, so that the blood within, or its coagula, are in actual contact with the spine, the wall of the aneurism merely closing in the diseased spot on each side. In this very common form the sac is generally oval, and communicates with the artery by a long oval opening. If the aneurism should come off from the side, it soon reaches the spine, when the same effects are produced, only then the absorption is more lateral, and the ribs may be involved. Even if the aneurism be formed on the anterior part of the aorta (which is not often), it may bulge over on one side, and affect the spine in the same way. Such aneurisms may cause death, as before mentioned, by opening in to the trachea, bronchi, or œsophagus; or they, not uncommonly, burst into the chest, producing sudden death; in some instances they penetrate the lung, as you lately saw, and, making their way through this organ, reach the parietes of the thorax. They also sometimes extend beneath the costal pleura, which thus gives them a false covering. If the parts near which they run are not actually laid open, we generally find these altered in form by the pressure, and thus we meet with a flattening of the œsophagus or bronchi, and very often disease in the lung; the immediate cause of death being pneumonia. Aneurism is more rarely met with at the lower part of the thoracic aorta, and may be of the fusiform or sacculated kind. Here is one showing an aneurism opposite the bifurcation of trachea, and another at the diaphragm. After leaving the arch of the aorta, the next most frequent place for aneurism is where the vessel passes through the diaphragm, so that sometimes a part of the tumour is seen in the chest and a part in the abdomen; or perhaps it is more frequent a little below this spot, and near where the cœliac axis comes off; the effects differ according as it is formed from the posterior or anterior part of the vessel. If from the back, it absorbs the vertebræ, as before seen; but if formed in front, a tumour projects anteriorly, and which involves in it very often the branches of the cœliac axis; these come off from the aneurism, or pass along its sides, and are compressed by it. In these cases the semi-lunar ganglia and cœliac plexus are often involved, and probably produce some of the symptoms. More rarely, aneurisms occur in the course of the branches of the abdominal aorta. Thus, here is an example of aneurism of a branch of *colica sinistra*, with another in the same case on the gastric, and two on the cystic arteries. Such aneurisms of small arteries are sometimes evidently due to embolism. Here is an aneurism on an arterial twig in the forearm, and one on a meningeal twig in the *dura mater* in the same case. All these examples are from two cases of ulcerative endocarditis, and, no doubt, were due to embolism. In this case also the femoral artery of a boy with ulcerative endocarditis shows a large aneurism also due to embolism. In cases of aneurism in young persons there has often been a history of acute rheumatism. It was so in a case of axillary aneurism in a girl under Sir W. Gull's care; also in a case of aneurism of mesenteric artery. In these rheumatic cases there is always a probability that rheumatic carditis and embolism may have been the cause of the aneurism; but, we may ask, does it happen that along with rheumatic endocarditis there may also be a rheumatic endarteritis, weakening the affected spot and leading to dilatation and aneurism? We have occasionally met with a patch of inflammatory softening of the aorta near, but not continuous with, aortic valves that were acutely inflamed from rheumatic fever, but in all instances but one there was reason to ascribe the aortitis to impact of vegetations attached to the valves. So that we know of but slight grounds for supposing that there is an arteritis directly rheumatic. We have before spoken generally of subacute arteritis.

In the middle-sized arteries of the limbs aneurisms are very common, and our shelves will show you the several varieties, and the effects of tying the vessel above them, and the large anastomotic vessels resulting. We have yet no specimen where a cure has been effected by pressure, but we apprehend the sac would resemble very much those hard tumours of the popliteal in which the femoral has been tied; for the object of the surgeon is so to lessen the circulation in the vessel as to cause coagulation in the sac. Here is a remarkable specimen where an aneurism of the iliac artery burst into the cæcum, causing death by hæmorrhage from the bowel.

Aneurisms within the cranium are frequent as compared with their rarity elsewhere in vessels of the same size; you might, perhaps, expect this, remembering how thin are the intra-cranial arteries. They often occur in young persons, a large proportion under the age of twenty-one. You here see specimens of aneurism of the basilar and cerebral arteries. If small, they may give rise to no symptoms until they burst, when the patient is suddenly seized with sanguineous apoplexy; if they be in the cerebral substance, softening may ensue; or they may produce local paralysis. We have met with a case of aneurism within the cavernous sinus which paralysed the motor nerves of the eye and then burst into the middle lobe of the brain, as in one of these cases where the tumour pressed on the third nerve, producing paralysis of the eye. Aneurisms occasionally are met with in the coronary vessels of the heart, and there is a specimen in St Thomas's where a number of small aneurisms exist upon them, giving them a beaded or varicose appearance.

A false aneurism is a sac filled with blood communicating with an artery, but the walls of the sac not formed from the vessel itself. It is most commonly *traumatic*, being produced when the artery has been punctured; as, for example, the femoral, by a knife or sharp-pointed instrument, when the blood escapes into the tissues around, forming a circumscribed pulsating tumour. This is a form of disease which you will hear much of in the surgical lectures, as there are very important points of treatment connected with it.

Aneurismal varix is where the artery has been punctured through a vein, so that the two communicate, a subject also fully treated of in the surgical lectures.

Varicose or *general dilatation of the arteries* is occasionally met with; one of the most remarkable is reported by Mr Adams, of the London Hospital, in which the patient had all his arteries, or at least those of his limbs, immensely dilated, being two or three times their natural size; and the consequence was a fatal hæmorrhage from an ulcer.

Dissecting aneurism arises when blood bursts through the inner coats of a blood-vessel, and passes down between its outer coats; it has generally been thought that this occurs between the external and middle coat, but Dr Peacock has stated that the middle coat itself is sometimes split. The affection arises from the more chronic form of arteritis, as before stated, the blood entering an atheromatous sac or passing behind a calcareous or otherwise affected part of the coat.

Sometimes you see remains of a former attack, in the presence of whitish fibrinous layers between the coats. In some cases the blood penetrates the internal diseased coat, and, after passing down a greater or less length of the vessel, re-enters by bursting again inwards through the coats. Under these circumstances recovery may ensue. A most remarkable example of this is given by Dr Fagge in the 'Medico-Chirurgical Transactions.' The proper coats of the arch of an old gentleman's aorta were torn through, and the blood passed down between the middle and outer coat on one side of the aorta through its whole length, reaching to the common iliac arteries, into which it again burst by a distinct opening on each side. In this remarkable case the patient lived some years after the rupture of the vessel, and the new channel took on so much the appearance of the interior of an artery that it was quite difficult to say which was the proper aorta and which the adventitious, for the latter had a perfect lining resembling exactly the inner coat of the artery, and having, like this, patches of fatty degeneration resembling ordinary atheroma.

Sometimes an aneurism treats the œsophagus in the same way in separating its coats. Thus, we have seen an aortic aneurism burst in between the muscular and mucous coat high up the œsophagus, and run down between these coats to burst into the stomach at its middle.

Aneurism by Anastomosis—Telangiectasis—Nævus.—Growths composed principally of blood-vessels, or of tissue communicating with the bloodvessels, may affect various parts, but especially the skin. They are for the most part congenital, though they may increase rapidly after birth, and occasionally, we think, they may commence at any period of life, especially after an injury. They have generally been divided into three classes, according as the capillary, venous, or arterial element prevails; and no doubt many varieties exist in this respect. Thus, sometimes a vascular growth may be seen to be full of venous blood occupying sinuses; at another, large arteries lead into it, by which a pulsation is imparted to it; and in other cases, as so often seen in nævi of young children, the vascularity is due to an increase of the smallest vessels, or capillaries. Besides these, many nævi consist of a new tissue, composed of cellular or membranous structure, forming spaces in which the blood is contained, this cavernous tissue communicating with the blood-vessels; whether this tissue is formed from the vessels, as the veins, or is a distinct formation which subsequently becomes vascular, we think is scarcely yet made out. We shall, when coming to Liver, be able to show you some specimens of cavernous tissue in that organ communicating with the veins. A change which nævi sometimes undergo is called the cystic degeneration; thus, after their existence for a certain time, a number of cysts form, and the vascularity is in great measure lost; it is indeed often a question for the surgeon, whether some of the cystic tumours of long standing which he removes have originally been nævi or not. Thus in this specimen, consisting of a mass of cysts now empty, which Mr Cock removed the other day from a boy's neck, there were large blood-vessels passing into it, and filling the spaces with blood, so that it nearly emptied itself, after removal. The growth appeared intimately connected with the blood-vessels of the part, but there was no unequivocal history of its having been preceded by a nævus.

Injuries.—*Rupture of Aorta.*—We have mentioned the occurrence of rupture of the vessels through disease of their coats, causing aneurisms, apoplexy, &c. But besides these there is a small class of cases in which the aorta suffers rupture, generally by reason of hypertrophy of the heart. The laceration is mostly transverse, but it has been seen to have a longitudinal direction; it usually occurs in the arch of the aorta. This accident will of course cause sudden death, but if the rupture spare the outer coat a dissecting aneurism may result, as in the remarkable case of Dr Fagge's, just alluded to.

Injuries to bloodvessels form a subject more proper to the surgical lectures, but you should notice carefully the liability of the great arteries to be torn through in fractures and dislocations, a very serious complication of such accidents; or the abdominal vessels may be burst in crushing injuries to the abdomen, it may be the renal or the mesenteric. Sometimes the aorta is pierced by instruments or lacerated by the rough contact of a fractured sternum. Such injuries are usually immediately fatal, but a small puncture may kill more slowly. Thus, a needle wound of the ascending aorta caused death in two hours. Sometimes the aorta is lacerated through injury in such a manner as will prove it to have been burst rather than torn by rough contact. In fracture of the sternum we have found a rupture of the aorta in the concavity of its arch, while the convexity which is towards the broken bone was not injured.

A very important example of injury to great blood-vessels, to which we would ask your best attention, is met with in some cases of tracheotomy. The end of the tube causes ulceration of the trachea, the ulcer spreads through its wall until the tube may reach the brachio-cephalic or common carotid artery producing immediately fatal hæmorrhage.

Morbid Growths.—*Tubercle* is not known to attack blood-vessels. *Cancer* never primarily affects the arteries. They are also very callous to the seductions of cancer when reaching their walls from other parts. It is remarkable for how long a time a great artery will resist all the operations of a growth of cancer outside it, and thus we often find a large mass of cancer, as, for example, in the mediastinum or the lumbar glands, with the aorta passing through its midst quite unaffected, and usually without compression or other alteration of its calibre. The veins, on the other hand, are generally implicated in the growth. Rarely, however, and in exceptional cases, the coats of the arteries may become involved, especially the thinner coats of the pulmonary artery in mediastinal cancer, but even then we have never seen the elastic coats changing to cancer like other tissues, but they are pressed in and wasted away by the cancer, and so a cancerous nodule be seen projecting into the interior. When, however, a large artery is near a cancerous sore in a sloughing state the vessel's wall is apt to give way along with other tissues in the sloughing process. This generally results from sloughing epithelial cancers. Thus such cancers in the pharynx or œsophagus may reach the carotid, or cancer of the œsophagus only may reach the aorta.

DISEASES OF THE VEINS

Malformation.—The various deviations from the normal standard of distribution of the veins will be pointed out in the anatomical lectures. Upon this heart you will see the left brachio-cephalic vein passing down by itself at the back of the organ, to enter the right auricle separately, instead of crossing in front to meet with its fellow. Here is a curious case; the right pulmonary vein gives off a long channel, which runs straight forward and ends in a cyst of the size of a plum, which lay adherent on the inner side of the front of the lung between it and the pericardium. It was full of a clot which was evidently of post-mortem formation.

In connection with this we will mention certain well-known curious blood-cysts that occur about the neck as well as in other parts. Their origin is obscure, but some have been traced into continuity with large veins (*e.g.* the femoral). Here is a large specimen which was removed and has been described by Mr Birkett, and which extended from the neck into the axilla. Some of these cysts have been clearly traced to the dilatation of the vessels in nævi. Others arise by hæmorrhage into simple cysts. These are thin-walled cysts with fluid blood in them, and must not be confused with those blood-clot tumours in the limbs which are so apt to prove malignant. The latter generally show clot of various date, and careful search will generally discover a patch of malignant growth in them whose vessels have given way to cause the hæmorrhage.

Inflammation of Veins; Phlebitis.—In considering inflammation of the arteries we found that acute inflammation of them is very rare, and that when reached by neighbouring inflammation they show very little disposition to adopt its activity, being, indeed, of remarkably callous nature, more resembling cartilage in this than any other tissue. Now, this is not quite the case with the veins, for although phlebitis once was thought to be more widely spread and important than it really is, this must not induce us to overlook the fact that acute inflammation of veins is a tolerably frequent and often a very serious occurrence.

When a vein is acutely inflamed it is always found full of clot undergoing changes. The amount of clot distends the vessel so as to make it feel both during life and after death like a hard cord. At the same time the wall of the vein is thick, and when cut into it appears as substantial as the wall of an artery. If the inflammation be very severe you find pus between the coats, perhaps in several layers.

Now, there can be no doubt that in many cases the clot found in the thick-walled and inflamed vein is truly blood-clot. But in other cases there must be equally no doubt that, along with the blood, inflammatory products are present until the clot may have all the microscopical characters of pus.

At the risk of being tedious we must shortly show you as well as we can the present position of the question of phlebitis, which is always one of the most important questions in pathology, and in some form or other, has exercised great influence on the views of pathologists at every period.

It will, we think, be best for us to put at once before you the intricacies of the subject with as little entanglement as possible. Puriform matter had long been known to occur in the veins, and was accepted as pus. Starting then from that time when *puriform* matter in the clots within inflamed veins was accepted as *purulent*, which was the first and natural view taken of it, we first meet with a denial of any such purulent nature of clots within veins by Goodsir and his followers. This denial was based on the microscopic appearance of the puriform matter, which was stated to be that of molecular detritus and not of pus. This denial had become universally prevalent, but in the last few years it is very gradually being admitted that the microscopic evidence of the non-purulent nature of this puriform matter is not so decisive as was until recently assumed, so that explanations are wanted. Virchow, for instance, and others, allow that a large number of pus-like corpuscles are present in the puriform matter, but they explain these pus-like corpuscles by pointing out that white blood cells are less destructible than red ones, so that when the red corpuscles in a clot have dissolved and been washed out of the clot, the white corpuscles still remain and appear as pus cells. But then sometimes there are far too many pus cells to be explained in this manner; hereupon Rindfleisch and others appeal to the adhesive property of the white blood cells, which induces them to cling to the surface of the clot or to penetrate within its spongy texture, fresh layers of clot forming then over the layer of such adherent white cells; when such a complex clot is supposed to soften, then it is thought that a sufficient explanation of the large number of pus-like cells is achieved. But we must remind ourselves here that the evidence has undergone a curious revolution from Goodsir, who bases the non-purulent nature of clots on *absence* of pus cells, to Rindfleisch and others, who explain away their *presence*.

In the meantime, however, the well-known experiment of Cohnheim, which you should all repeat, shows that the pus in the inflaming mesentery or cornea of a frog is nothing else than out-wandered white blood cells; thus, white cells out of place claim to be pus-cells. Here, then, we find another awkward complication thrust on the question, especially since Weber has caused the white cells of the blood to take up cinnabar after a clot has first been formed, and then proved that these cinnabar-bearing cells find their way into the clot after its formation; such white cells wandering into the clot are as much pus as the white cells are when wandering out of the vessels in the mesentery. Thus, on the view of suppuration, which is based on Cohnheim's experiments, it is difficult to distinguish the pus-like cell-bearing matter in such clots from true pus; in fact, on that view it is pus.

We take you over all this rather intricate ground because it is very necessary to be clear about the nature of the processes within the vessels; for, although the details of interpretation may not be altogether agreed on in the most modern times, yet it has been certainly shown that the formation of these clots and their effects on the vessel they form in, and the effects of their removal to remote parts, play a supremely important part in many grave and fatal pathological processes.

We shall endeavour to show you that some clots contain pus and some do not, and for this purpose we will leave the theoretical consideration of the matter and consider the different anatomical forms in which phlebitis is met. As in other inflammations these are partly different degrees of severity, partly different kinds due to unknown differences in their causes.

Pus in the veins; suppurative phlebitis.—The channel of a vein sometimes forms part of an abscess; thus, an abscess of the liver bursts into a hepatic vein and the pus passes up the vein some distance, being shut off by a fibrinous layer from mixture with the blood beyond; or the pus may pass up along the vein further and reach the *cava* and go up beyond and enter the right auricle of the heart; or an abscess about the hip will so open the femoral vein that a part of its channel extending up into the iliac vein shall be like an open offset from the cavity of the abscess. The presence of pus within the vein is in such cases indisputable, the wall of the vein is found to be altered by thickening and formation of granulations on the surface, or by pus in layers in its substance. But we have found the veins near suppurative inflammations containing true pus when there was no continuity of their interior with an abscess. This is, indeed, frequently the case with the veins of an ill-conditioned stump, after death by pyæmia following amputation or acute necrosis, compound fracture, or burn. We have seen also in such cases a clot, containing what microscopically was pus, lodged in the pocket of a valve in the femoral vein at a distance above another purulent clot.

In a case of pyæmia from a bubo there was a pus-clot which went up the iliac vein from the femoral and then extended down the *inferior hæmorrhoidal* veins to an inflamed pile. This clot was full of pus and there were ante-mortem polypi in the right auricle also showing pus.

Another grave and important kind of case is where there is purulent clot in the sinuses of the dura mater; this is generally set up by caries of the neighbouring bones, especially the bones of the ear, or else it may be caused by traumatic ostitis with or without fracture, but it is not exclusively due to disease or injury of the bones, it may arise through extension of phlebitis along the facial veins, a terrible risk incurred in cases of malignant pustule as we have several times verified, also occasionally met with in cases of ulceration in the nasal fossæ, &c. Again, in operations about the uterus or bladder the pelvic veins may show truly purulent clot extending up their ramifications as far as the iliac veins. This is the best established cause of phlegmasia dolens in lying-in women. In all these cases the inflammation extends from the minuter radicles of the veins up the branches to the trunk; but sometimes a vein suppurates through injury, as after phlebotomy, or in the interesting class of cases of inflammation of the umbilical vein in new-born infants; in these suppuration may extend into the interior of the body, producing abscess of the liver or peritonitis.

The means of conveying the inflammation in all these examples is the clot formed in the blood, which clot extends up from twig to branch and from branch to trunk. This clot, poisoned with the diseased juices from the wound and purulent with in-wandered white blood cells, inflames the wall as it spreads. We cannot join with those observers who treat of the clot as if it were always simple fibrin undergoing only ordinary changes of simple decomposition. These observers do not give weight to the truly purulent microscopic character of the clot, they follow Goodsir's account of thrombus which Virchow adopted so implicitly; but Goodsir's account of soft thrombus, as only detritus, applies only to thrombus within the heart and non-inflammatory or sub-inflammatory thrombus generally; while we are now bound to admit also an inflammatory thrombus which contains pus. Whether this pus truly arises by wandering in of white cells out of the blood as in pus-formation outside the vessels by the wandering out of these cells, or whether it be due to some process of pus-formation not yet cleared up, is a question in the explanation of the presence of pus; but there can be no question as to the fact of its presence in the cases we are speaking of. The determination of some authors to regard all puriform matters within the clots in inflamed veins as only disintegrated clot, amounts almost to prejudice; you will see authors in compiling take the original describer's account of purulent clot in the vein and explain it away as puriform. Thus, Dusch, in his admirable 'Monograph on Thrombosis of the Cerebral Sinuses,' assumes that all puriform products described as pus by their actual observers were mere softened thrombi, when, for instance, he quotes a case from Pitha he turns the word "purulent" to "puriform;" but our own observation agrees with the description of Pitha, for we have found, histologically, perfect pus in the sinuses of the dura mater in a case similar to that quoted. Dusch's paper, however, draws an important distinction as to the clots in the cerebral sinuses, which we will show you extends throughout the whole venous system, namely, the distinction of clots caused by mechanical stasis, and clots due to the neighbourhood of suppurative inflammations extending to the walls of the veins,

Primary suppurative phlebitis is, fortunately, very rare; so rare is it that it is constantly ignored. It is, perhaps, most certainly recognisable in the portal vein. Many cases of suppurative "pylephlebitis" have been recorded; we have met with two examples. Under Liver we shall speak of this again. We have met with suppurative pelvic phlebitis in gonorrhœa without abrasion of the mucous membrane, and a similar case occurred some years ago under Dr Habershon's care. Both these cases ended in fatal pyæmia.

Adhesive inflammation of veins, and chronic or laminated thrombosis.—Plastic inflammation or "sub-inflammation" of the veins is of frequent occurrence. You occasionally find a vein, which has been pressed flat by a neighbouring tumour, closed up by adhesion of its apposed surfaces, &c. But plastic phlebitis is generally a consequence of thrombosis in states of low pyrexia. (*Thrombosis* is a coagulation of the blood *in sitû.* A *thrombus* is a clot formed *in sitû.*) We will remind you again of the causes of coagulation of blood in the vessels. They are—1. Stoppage or extreme slowness of the current. This is one effect of roughness of the containing surface as the blood is slowed in its irregularities. 2. Contact with an abnormal surface, such as a surface deficient in vitality or altered in its vitality. 3. Increase of fibrin in the blood, either an absolute increase or an increase of the coagulating tendency of the fibrin, such as is observed especially in low febrile states and cachectic inflammations in pregnancy, &c. By a little consideration you will perceive that in bedridden persons near death, or in those excessively reduced by fever, &c., the first two conditions are likely to arise in some portions of the venous system earlier than in others, so that the blood will tend to coagulate in some parts while it circulates in others. We will shortly mention the chief seats of such occurrence. The weight of the body falls especially on the buttocks, and the gluteal, circumflex, and other veins of that part are much impeded, so that the stream in these branches may stay and the blood coagulate, the clot extending from small branches into larger until it protrudes into the main femoral or iliac veins where it is increased in size by layers of clot, until it may quite stop up the femoral vein. While the clot is in the branch in which it arose it is called an *autochthonous* thrombus; where it extends into the main trunk it is said to be a *produced* thrombus. When there, it is exposed to the whole current from the lower limb, and it is liable to be broken off and carried on in the current until it is cast into the pulmonary artery as an *embolus;* or before it is so carried away, the blood in the femoral and iliac veins for several inches may be coagulated and added to it, forming a long mass which has a bluntly-pointed end towards the heart, and bears on its sides moulds of the valves it lay against. We have known such a clot extend all the way up the *vena cava*, and present its blunt point within the right auricle. If such a large clot moves up into the pulmonary artery the result is speedy or even immediate death. Often the clot commences in the pocket of a valve. In examining such a clot you find the older portions partly decolorised and washed clean of its dissolved red corpuscles, while the newer part is mingled brownish or purplish-red, according to its age. If it have lasted long in its place it may be found softened down to a fluid of creamy consistence and having a dirty brown or purple colour often compared to that of wine-lees. This softened pulp in non-inflammatory cases shows microscopically little more than granular detritus, but as we have already said, in grave inflammatory cases true pus may be found and pyæmia coexist.

If the clot, instead of moving to embolise the pulmonary artery, stay in its place it may disintegrate and pass away, or it may go through various changes of a developmental kind whose end is to reopen the channel of the vein. It is said, especially by Mr Gay, that the exterior of the clot may harden and unite with the thickened wall of the vessel and the centre of the clot soften and open up as a channel, but we have not verified or seen proof of this. Or, again, after a long period during which the clot has adhered firmly to the wall of the vessel, both the clot and the vessel may dwindle away to a small fibrous cord. Thus the vena cava may become obliterated through disease in its neighbourhood in which it becomes involved, so that at last it is found contracted or quite closed. In such a case the superficial veins become immensely distended.

Another part of the venous system which shows great liability to stagnation and coagulation is the pelvic plexus of veins. The functions of the generative organs require a variable state of circulation, and this tends to induce a widening of their venous system beyond what is necessary in ordinary states. Under obstructive conditions this renders stagnation and coagulation frequent. Thus, clotting may occur in the *vena dorsalis penis*, or prostatic plexus; coagulation from this source also may extend up into the iliac veins, but it does so more rarely than that from the circumflex veins. The clots in these veins not infrequently persist and undergo calcareous transformation, forming phleboliths.

Again, the sinuses of the cranium, in cases of marasmus, form another favourite seat of coagulation. The delay is here occasioned by the wasting of the brain and consequent tendency to detain blood to fill the vacuum that would arise in the closed skull. We have occasionally seen thrombus of the cerebral sinuses in children with marasmus from this cause. Dusch has drawn attention to these cases, and points out the distinction between them and thrombus from diseased bone, in the position of the clots, which are symmetrical in the simple cases due to stagnation, but are found close in the neighbourhood of the disease when secondary to local inflammation. Another not infrequent position for thrombus is in unnaturally dilated veins, such as saphenous varices or hæmorrhoids, and another very important and not infrequent position is in the portal vein in severe cirrhosis of the liver. Again, thrombus is far from rare in the renal veins. We have met with it under these conditions:—1st, in infants who have died of diarrhœa and wasting; 2nd, in the large white kidney of Bright's disease, especially when lardaceous; and, 3rd, in cases of severe injuries to the lumbar region ('Guy's Hospital Reports,' 1868). We have seen once general phlebitic thrombosis in the veins of the limbs in Bright's disease; it was a case of granular kidney.

Now what we wish especially, even if repeatedly, to impress upon you is the variety of results which must arise when to the simple stagnation and low vitality there is added an inflammatory febrile or cachectic state of the blood; certainly there will then be from this cause a greater liability to the coagulation through the accompanying increase of the fibrin in the blood; and thus it is that you most frequently find the thrombi in these patients. For example, in phthisis these clots are so very frequent that it is quite the general rule to find them, unless the patient is carried off by some intercurrent affection; also, it is thus that in heart-disease or in Bright's disease the clots form usually most in those with whom a pneumonia pleurisy, or peritonitis has complicated the later stages of the disorder. In some cases this last cause of thrombosis, the altered state of the blood, appears to be the more obvious and primary cause of the change, though the situations of its occurrence still are the same as we have mentioned; so that, evidently, stagnation has its share. Thus, after fevers, especially enteric, but occasionally too after typhus or relapsing fevers, you meet with thrombus in the femoral vessels, also we have met twice with a primary or idiopathic thrombosis in that situation.

But the point of main interest is the variation of local effect of the thrombus according to the character of the general disease present. When there is a generally inflammatory or impure febrile state of the system then the vein wall becomes inflamed, and the inflammation is more obvious and its results more grave in proportion as the general fever runs high. For instance, the clot formed by the blood poisoned from the uterine sore after parturition, or by the disordered blood of fever, produces a clot in the usual situation of stagnation in the femoral vein, which behaves very differently to the clot that forms in the last stages of a cancer or other simply wasting disease. Where the clot in the febrile case rests against the vein, its wall inflames. Why this inflammation? Some set it down to simple changes in the thrombus due to decomposition; but the clot in cancer may often be proved to be much older, and yet such simple thrombus as you find in cancer cases will sometimes remain long in the veins without even becoming adherent to their walls; so that is not the effect of duration and its consequent changes. We think the cause is in the quality of the thrombus due to the quality of the blood that forms it. The blood is impure; where it circulates freely its impurity is always being partly removed by the depurating organs, but where clotted the clot becomes a reservoir for the impurities in which they can accumulate. The clot is unrelieved by the excretory changes which the blood would undergo in passing through the lung, liver, &c., and hence becomes poisonous and irritates and inflames the vein, thus totally obstructing it by the swelling of its wall, and, perhaps, producing inflammation around, which reaches and obstructs the lymphatics, so causing more or less of *phlegmasia alba dolens*, which is the name given when the obstruction in these febrile and inflammatory cases reaches its height.

On the other hand, when there is no more than simple stagnation, the blood not being morbid, the clot will remain in the vein, and there will be scarcely any inflammatory result, so that the thrombus may not produce any phlebitis or even adhere to the wall of the vessel. Such cases are frequent enough, in cases of dropsy, which die without inflammation in cancer, &c.

An intermediate class is found in some cases of phthisis, &c., where a simple inflammatory pyrexia coexists with the thrombosis. In these cases some slight inflammation of the vessel and adhesion of the clot is usually met with.

We have seen thrombosis of the veins of the upper limbs in rheumatic heart disease, but have not as yet been able to explain why the coagulation occurs there instead of, as usual, in the lower limbs.

Plastic phlebitis is occasionally met with in a primary form, we have seen it, especially in elderly people, with varicose veins. This state has been said to be accompanied by saccharine urine, but we have not yet noticed this connection. The superficial veins of the forearm have thus inflamed without evident cause, also the veins of the legs, especially if varicose. We have known such simple primary phlebitis produce fatal embolism.

We would draw your attention to the curious absence of dropsy in some cases of extreme emaciation when the whole venous system of the lower extremities, *e. g.* one or both common iliac veins, are quite full of thrombus, as far as we could make out, entirely adherent all around the vein.

Embolism.—It will be convenient now to consider the effects of the movement of clots from the site of their original formation, and their conveyance onwards by the blood stream until they reach new lodgment in arteries too small to allow them to pass. A clot which is so moved is called an embolus, and the process of its impaction in the artery is called embolism.

Being fresh from the consideration of the various kinds of clot or "thrombus" found in veins, we shall be now able to inquire what are the effects of embolism of the arteries with these several kinds of clots. First we will consider the effects of the mere stoppage of the current by the mechanical effect of the embolus. Then we will follow out the results that supervene on account of the irritative qualities of the clot.

The mechanical effects of the embolus are not quite what you would at first expect. One naturally thinks that if the artery of a part is stopped up the blood supply will be shut off, and the part become bloodless. But, when a part of an organ is embolised, the effect is that its vessels become congested—that is, if time be allowed; for in some cases, as when the pulmonary trunk is plugged, death occurs at once through the anæmia beyond; and in embolism of the femoral, the limb may in like manner die and become gangrenous. To understand the congestion that arises from more limited embolism, you must remember that the vessels are not passive tubes, but that they keep their size by reason of their vital contractile power, and that they dilate when that power is lost or diminished. Next we must remember that the whole blood system is in a state of tension, so that when a vessel is divided the blood gushes out, or if a tube is put into the vessel the blood runs into the tube and fills it to a certain height. Recalling these facts, we are ready to see that where an artery is plugged its tissue beyond the plug being deprived of nourishment will lose its contractile power and give way, offering no resistance to dilatation; and meanwhile the tension of the whole blood forces some into this dilated space, as it would into a glass tube or other line of escapement—in short, there is a sort of hæmorrhagic escapement of blood into the starved, paralysed, and dilated vessels. But, you may ask, why is it that when the blood so enters anew into the vessel, this does not recover itself and contract? Here, again, you must observe that the blood that thus has entered has no longer got the force of the heart playing on it directly, so that it does not circulate, until it protrudes into the main femoral or iliac veins where it is increased in size by layers of clot, until it may quite stop up the femoral vein. While the clot is in the branch in which it arose it is called an *autochthonous* thrombus; where it extends into the main trunk it is said to be a *produced* thrombus. When there, it is exposed to the whole current from the lower limb, and it is liable to be broken off and carried on in the current until it is cast into the pulmonary artery as an *embolus;* or before it is so carried away, the blood in the femoral and iliac veins for several inches may be coagulated and added to it, forming a long mass which has a bluntly-pointed end towards the heart, and bears on its sides moulds of the valves it lay against. We have known such a clot extend all the way up the *vena cava*, and present its blunt point within the right auricle. If such a large clot moves up into the pulmonary artery the result is speedy or even immediate death. Often the clot commences in the pocket of a valve. In examining such a clot you find the older portions partly decolorised and washed clean of its dissolved red corpuscles, while the newer part is mingled brownish or purplish-red, according to its age. If it have lasted long in its place it may be found softened down to a fluid of creamy consistence and having a dirty brown or purple colour often compared to that of wine-lees. This softened pulp in non-inflammatory cases shows microscopically little more than granular detritus, but as we have already said, in grave inflammatory cases true pus may be found and pyæmia coexist.

If the clot, instead of moving to embolise the pulmonary artery, stay in its place it may disintegrate and pass away, or it may go through various changes of a developmental kind whose end is to reopen the channel of the vein. It is said, especially by Mr Gay, that the exterior of the clot may harden and unite with the thickened wall of the vessel and the centre of the clot soften and open up as a channel, but we have not verified or seen proof of this. Or, again, after a long period during which the clot has adhered firmly to the wall of the vessel, both the clot and the vessel may dwindle away to a small fibrous cord. Thus the vena cava may become obliterated through disease in its neighbourhood in which it becomes involved, so that at last it is found contracted or quite closed. In such a case the superficial veins become immensely distended.

Another part of the venous system which shows great liability to stagnation and coagulation is the pelvic plexus of veins. The functions of the generative organs require a variable state of circulation, and this tends to induce a widening of their venous system beyond what is necessary in ordinary states. Under obstructive conditions this renders stagnation and coagulation frequent. Thus, clotting may occur in the *vena dorsalis penis*, or prostatic plexus; coagulation from this source also may extend up into the iliac veins, but it does so more rarely than that from the circumflex veins. The clots in these veins not infrequently persist and undergo calcareous transformation, forming phleboliths.

Again, the sinuses of the cranium, in cases of marasmus, form another favourite seat of coagulation. The delay is here occasioned by the wasting of the brain and consequent tendency to detain blood to fill the vacuum that would arise in the closed skull. We have occasionally seen thrombus of the cerebral sinuses in children with marasmus from this cause. Dusch has drawn attention to these cases, and points out the distinction between them and thrombus from diseased bone, in the position of the clots, which are symmetrical in the simple cases due to stagnation, but are found close in the neighbourhood of the disease when secondary to local inflammation. Another not infrequent position for thrombus is in unnaturally dilated veins, such as saphenous varices or hæmorrhoids, and another very important and not infrequent position is in the portal vein in severe cirrhosis of the liver. Again, thrombus is far from rare in the renal veins. We have met with it under these conditions:—1st, in infants who have died of diarrhœa and wasting; 2nd, in the large white kidney of Bright's disease, especially when lardaceous; and, 3rd, in cases of severe injuries to the lumbar region ('Guy's Hospital Reports,' 1868). We have seen once general phlebitic thrombosis in the veins of the limbs in Bright's disease; it was a case of granular kidney.

Now what we wish especially, even if repeatedly, to impress upon you is the variety of results which must arise when to the simple stagnation and low vitality there is added an inflammatory febrile or cachectic state of the blood; certainly there will then be from this cause a greater liability to the coagulation through the accompanying increase of the fibrin in the blood; and thus it is that you most frequently find the thrombi in these patients. For example, in phthisis these clots are so very frequent that it is quite the general rule to find them, unless the patient is carried off by some intercurrent affection; also, it is thus that in heart-disease or in Bright's disease the clots form usually most in those with whom a pneumonia pleurisy, or peritonitis has complicated the later stages of the disorder. In some cases this last cause of thrombosis, the altered state of the blood, appears to be the more obvious and primary cause of the change, though the situations of its occurrence still are the same as we have mentioned; so that, evidently, stagnation has its share. Thus, after fevers, especially enteric, but occasionally too after typhus or relapsing fevers, you meet with thrombus in the femoral vessels, also we have met twice with a primary or idiopathic thrombosis in that situation.

But the point of main interest is the variation of local effect of the thrombus according to the character of the general disease present. When there is a generally inflammatory or impure febrile state of the system then the vein wall becomes inflamed, and the inflammation is more obvious and its results more grave in proportion as the general fever runs high. For instance, the clot formed by the blood poisoned from the uterine sore after parturition, or by the disordered blood of fever, produces a clot in the usual situation of stagnation in the femoral vein, which behaves very differently to the clot that forms in the last stages of a cancer or other simply wasting disease. Where the clot in the febrile case rests against the vein, its wall inflames. Why this inflammation? Some set it down to simple changes in the thrombus due to decomposition; but the clot in cancer may often be proved to be much older, and yet such simple thrombus as you find in cancer cases will sometimes remain long in the veins without even becoming adherent to their walls; so that is not the effect of duration and its consequent changes. We think the cause is in the quality of the thrombus due to the quality of the blood that forms it. The blood is impure; where it circulates freely its impurity is always being partly removed by the depurating organs, but where clotted the clot becomes a reservoir for the impurities in which they can accumulate. The clot is unrelieved by the excretory changes which the blood would undergo in passing through the lung, liver, &c., and hence becomes poisonous and irritates and inflames the vein, thus totally obstructing it by the swelling of its wall, and, perhaps, producing inflammation around, which reaches and obstructs the lymphatics, so causing more or less of *phlegmasia alba dolens*, which is the name given when the obstruction in these febrile and inflammatory cases reaches its height.

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Varicose Veins.—A varicose condition of veins is very common in the lower extremities, known by their enlargement and serpentine course. The vessels, when examined, are found to be very much thickened in their coats, which may be as thick as those of arteries, sometimes they are dilated into large spaces reminding of aneurisms of the arteries, but although they burst sometimes like aneurisms yet the pathological course of the two affections is very different. Veins tend to elongate and become tortuous, arteries to widen and dilate into great sacs; but we have here a large aneurism of the portal vein, which formed a great blood tumour behind the liver. The portal vein, however, is, in many respects, like an artery rather than a vein. When the veins are about to dilate and become varicose the widening is found first just above the valves, then the valves give way and the vessel stretches, lengthening more than it widens, so that it is thrown into curves, which increase in number while they dilate in some parts more than in others. The cause of this affection is not positively known. It appears very certain that pressure on the trunk of a vein will produce it, and therefore various surmises have been made as to the cause of the pressure; thus, pregnancy is constantly spoken of; but then, it must be remembered, that the disease is more common in men; also, constipation is mentioned, but of this there is no proof. Further investigations are certainly required to determine with accuracy its cause, especially, we think, careful dissection of the saphenous opening and the neighbouring glands. In cases of great varicosity of the crural veins the amount of blood withdrawn into the legs on assuming the erect posture, and, of course, also thrown back in the horizontal position, has to be estimated in considering the causes of apoplexy and softening of the brain or the effects of any coexistent dilatation of the heart.

Hæmorrhoids.—Formerly these were considered to be varicose veins of the rectum, but this is rarely the case; enlarged veins may be found in external piles; but you will be surprised to see how much fibro-cellular substance is present in an ordinary pile, though it is highly vascular and corresponding mostly to the fold of mucous membrane seen within the bowel. Very often piles have a true cavernous structure, being composed of cells which are filled with blood and communicate freely with the hæmorrhoidal veins, their inner surface being continuous with the lining membrane of the vessels. They probably are formed in the first place from the veins, which distend and then break through into each other by destructive pressure on the intervening tissue.

Varicocele is the term applied to varicose condition of the veins of the testes. The explanation of its more frequent occurrence on the left side, is the greater dependency of the testis, and the entry of the left spermatic vein into the renal vein. Not long ago we met with a case where the arteries were varicose, twisted together like a mass of worms, distended and bony.

Morbid Growths.—*Tubercle* is almost unknown in the veins.

Cancer does not occur primarily, but the veins may be involved in the disease. Thus, in those large masses of cancer affecting the lumbar glands, so often found in the abdomen or mediastinum, the coats of the vena cava may become implicated, and sometimes small sprouting fungous growths may be seen springing from the interior; or the vena cava may be utterly lost in the mass of a cancer.

DISEASES OF THE LYMPHATIC VESSELS

We will not occupy much of your time with this subject, which is almost entirely clinical; for *inflammation* of the lymphatic or absorbent vessels is best seen in the living subject, as after death the redness subsides, and anatomy throws little light on the early stages of the affection. When filled with pus, the vessels may be recognised by their knotted or varicose appearance; they may so be sometimes seen in the broad ligament passing up to their respective glands in cases of puerperal fever.

The most important affection of the lymphatics and lacteals is obstruction. This is not at all uncommon in tubercular disease of the mesenteric glands; you will see here many specimens of it. These vessels, of very irregular shape and distended, are seen coursing over the intestine and passing along the mesentery to the glands. In some cases, where their contents are hard, it is probable that some tuberculous matter has penetrated them. This appeared to be the case in this preparation, it is better seen in the drawing. The lacteals may constantly be found filled with chyle, without any obstruction being present, if the person die or be killed during the process of digestion. Sometimes, owing to obstruction of a duct, the chyle will collect and form a cyst, and thus small milk-white cysts may sometimes be found on the mesentery. In one very remarkable instance, which we saw here some years ago, such a cyst had burst, and the abdomen contained several pints of chyle resembling milk. Several similar cases of chylous fluid in the abdomen have been recorded.

Some cases have occurred in which great varicose distension of the lymphatics of the thigh or trunk has followed on some abscess, or other more or less obvious cause of obstruction. In such a case the lymphatics may form prominent cyst-like knots, puncture or rupture of which may yield pints of lymph.

We have recently met with several cases which make it probable that an obstruction to the course of the lymph stream, through old glandular disease, scrofulous or otherwise, may weaken the tissue of the part obstructed, just as bad drainage spoils the soil and its produce. The evidence of this was the occurrence of spontaneous low or ulcerative disease in such instances, limited to parts whose lymphatics led to obstructed glands, while the lymphatics themselves were full of pus. These occurrences we have found in the intestines from cretaceous degeneration of the mesenteric glands, and in the lungs and pleura from similar old disease of the bronchial glands.

The thoracic duct is liable to obstruction at either end: if at its lower part it receives no chyle, and if at its upper part it becomes distended; but as no great nutriment can enter the blood in either case marasmus ensues. In tuberculous mesenteric disease this inanition occurs; but one of the simplest and most severe cases was a patient from whom this specimen was removed; owing to some induration of the glands and surrounding cellular tissue in the left side of the neck, the termination of the thoracic duct had become obstructed; the consequence was a dilatation of the duct, as you see here, and the most wretched emaciation of the patient that it was possible to witness. In a case of ophthalmic goitre, where the end of the thyroid gland passed deeply into the thorax, pressure by it on the end of the thoracic duct seemed to be the cause of extreme emaciation.

Cancer.—Besides the conveyance of cancer by the lymph *stream* to the lymph glands, cancer also is sometimes conveyed along the lymphatics so as to appear in knots in their walls. Mr Birkett has placed in the museum some beautiful dissections of cancer of the breast showing this. Kœster believes himself to have proved that epithelial cancer spreads itself in the seat of its origin along the capillary lymphatics and takes its peculiar tubular forms from these.

DISEASES OF THE LYMPHATIC GLANDS

Malformation.—We would here mention to you an interesting, though theoretical, view of scrofula, which regards the characteristic glandular swellings of that disease as due to an original imperfection of the lymphatic glands, so that they are less permeable than natural, or liable to swell irritably, or otherwise to impede the flow through them on trifling accounts. You will remember how variable the glands are in their number, size, and mode of aggregation as you knew them in the dissecting room. A full consideration of these variations and their effects is still wanted.

Inflammation.—Inflammation of the glands is almost invariably due to irritating lymph from a disease in the part the lymph comes from; indeed, severe inflammation anywhere is apt to induce some sympathetic irritation of the corresponding lymphatic glands. Thus, in pneumonia you constantly find the bronchial and mediastinal glands very large, and pink or red in colour; but we should occupy too much of your time in detailing instances of this, which your experience will soon furnish in plenty. If the inflammation be of a "benignant" or simple nature the glandular irritation subsides with it and commonly is not noticed; on the other hand, if the inflammation be specifically poisoned, as in necrogenic pustules on the hands from dissection, or in soft chancre, then the swollen gland is apt to pass into a state of suppuration. It is curious and interesting to notice that some malignant inflammations have so great a tendency to attack the glands while others may leave these alone and spend their violence on the veins. We have twice seen inflammation from a malignant pustule or carbuncle of the face extend to the sinuses of the *dura mater,* and yet not implicate the glands at all. The implication of the glands in Oriental plague must be remembered, they play a very important part in its history under the title of buboes, and the facts observed prove that glands *may* be primarily affected in fevers.

Sometimes we have met a great abscess about the pelvic fascia, apparently originating in the glands from acute inflammation.

The minute anatomy of an inflamed gland is very simple, the small blood-vessels are distended with blood and the tissue is soft, showing vastly more of the lymph cells and less of the fibrillar network than is natural in the gland. But no question is more difficult to answer than that which asks how this simple change arises. Is it by wandering of lymph cells out of the vessels? Is it by multiplication of the lymph cells already formed? Is it by proliferation of the stellate cells of the meshwork? Each of these views has analogy in its favour. The most detailed descriptions, those of Billroth, favour the last view. We have seen these stellate cells containing many nuclei as if multiplying, but this condition was not so general as to persuade us that the great increase of lymph cells arose so. We rather think this action of the stellate cells of the stroma is connected with the increase of stroma and consequent induration which supervenes when the inflammation becomes chronic. It is most evident in *chronic inflammation*, which, in the glands, as elsewhere, differs anatomically from acute inflammation in producing increase of the fibrous stroma and comparative wasting of the cellular elements. We could not tell how the greatly increased number of lymph cells arose.

Particular glands are affected under special circumstances, generally from disease in the parts they receive lymph from, and thus the *mesenteric* glands are peculiarly affected in *enteric* fever. In this disease, associated with a peculiar deposit in Peyer's glands, the neighbouring mesenteric glands are also affected; these are very much enlarged, and, when cut open, are found red and soft. We have sometimes seen both the glands and the lymphatics full of effused blood in grave cases. The microscope shows the material to consist of a substance very rich in cells but having, on the whole, a close resemblance to the natural tissue of the lymphatic glands, so that it has hence been classed by Virchow with lymphoma (see Plate VI). It sometimes is so much like the soft fleshy growths that it was described as encephaloid cancer, a view that it is not very remote from that of Virchow's, since fast growing lymphomas form the most characteristic encephaloid cancer. But it is now considered, together with the formation in the intestine, to be peculiar to enterica, and hence it is called the typhous or typhoid deposit.

It has been the subject of question whether this change in the glands in enterica is due to the disease in the intestine or is an independent affection of the glands. Our own experience leads us to think that it is certainly secondary; for, on the whole, it bears a close and direct proportion to the extent of the intestinal mischief, and it affects the glands that correspond to the diseased parts of the intestine. We have no experience of the enlargement of the bronchial glands, which some say belongs specially to typhoid fever. When they were enlarged at all in our cases there was always pneumonia present, and this, as we have already said, will of itself cause enlargement of those glands.

In *scarlet fever* the glands of the neck are sometimes very greatly swollen. The circumstances, in relation to the fauces and the anatomy of these glands, are identical with those of the mesenteric glands in typhoid (see Plate VI). Sometimes these glandular swellings suppurate or slough and the consequences may be frightful; thus, we have seen the whole of the deep tissues of the neck lying as if dissected from ear to ear, the hyoid bone lying quite bare on one side.

Syphilitic disease of the glands.—The indolent bubo of syphilis and its contrast with the suppurating bubo from non-infectious sores you will hear enough of in the surgical classes. We would only advise you not to be guided too absolutely by this distinction, for we have known sometimes syphilitic disease to follow suppurating bubo; some carry the distinction of Ricord so far that we have heard a history of suppurating bubo given from the Chair of the Pathological Society as evidence that the patient had not had syphilis! We mention this only as showing how far a dominant idea not founded on an observer's own experience may carry him; but, of course, a man who has got one contagious sore is, at least, known to be the sort of person to run the risk of the other. Again, besides our own observation, we have been told by Mr Cock that he has certainly seen syphilis arise from soft chancre. Explanations of this by coexistence of the two poisons, &c., may be ingenious, but are not so important or clear that they should lead you to forget that there is something to be explained before we can accept it as a universal rule that soft chancres with suppurating bubo never cause syphilis. The minute anatomy of the syphilitic bubo has nothing in it characteristic; there is an increase of the lymphatic cells throughout the gland, as well as often an increase of the trabecular tissue, such glands do not usually tend to become caseous like scrofulous glands. You sometimes find the glands implicated in late syphilitic gummatous masses in the mediastinum, neck, &c., but the disease in them has always appeared to be secondary in our examples of these cases.

Morbid Growths.—*Hypertrophy.*—We have already mentioned the extreme liability of the lymphatic glands to swell, and given you instances of special diseases in which these swellings reach a large size, as in pneumonia, enterica, &c. Lymph-gland structure is so like some simple inflammatory products that the same description would roughly serve for both, we could say, namely, that either shows lymphoid cells lying in the meshes of a fibrillar material. This simple structure of the lymph glands we must ask you to take good notice of. If you consider it more precisely you will find that the meshwork of a lymphatic gland has, perhaps, two special features; first, in having stellate cells at intervals connected with it, and, second, in having a concentric disposition around the capillary vessels that supply the gland, the meshwork being separated from the vessel by an interval, which is the lymph path; the lymph-cells lie in small clusters in the meshes of the network. The stellate cells and the concentric arrangement are relied on in identifying lymph-gland structure, but these characters are certainly found well marked in tubercles, especially the large tubercles of the brain, and we have found them both in common inflammatory products.

If you duly notice how like lymph-gland tissue, which many regard as a permanently embryonic form of areolar tissue, is to inflammatory products, you will be ready for a difficulty in interpretation of the enlargements of these glands, for new matter in the gland due to inflammation will have the same structure as the gland itself. There is positively no difference whatever between the normal lymph cells of the gland and the exudation cells produced by inflammation. Thus an inflammatory enlargement is structurally a hypertrophy of the gland, and we have to distinguish the two conditions by their circumstances.

When in the absence of inflammatory cause the glands are enlarged without any alteration of their natural structure they are said to be *simply hypertrophied*. But simple hypertrophy of the glands is an unstable condition and tends to pass into caseous degeneration so as to become "scrofulous," or else it gradually takes on the size and relations of tumour so as to become "lymphadenoma." It is clinically convenient to call large glands "hypertrophied" when they are persistent and do not either suppurate or swell to great tumours. But there is no anatomical basis of distinction between the state of the glands in simple enlargement and that found in Hodgkin's disease, only that in the more moderate enlargement of simple hypertrophy the gland tissue is less altered than in Hodgkin's disease, and yet authors describe indurated as well as soft forms of hypertrophy. When this is allowed there is no distinction of hypertrophy from lymphadenoma, except in the circumstances and size of the growths.

General lymphadenoma (lymphosarcoma, Virchow; Adenie, Trousseau) Hodgkin's disease.—There are some cases of glandular enlargement associated with anæmia, which are not infrequent, and which present so close a mutual resemblance clinically that it is convenient or even necessary to have a clinical name for them. We use the name Hodgkin's disease for this *clinically* recognisable state in justice to Dr Hodgkin who first drew attention to the peculiarities these cases present. Of late a great share of attention has been given to the microscopical detail of their structure and its kinship to other states, but this should not draw us off from that clinical identity of the cases which is recognised under the name Hodgkin's disease. It is very important to clearly see the relation between Hodgkin's disease and lymphadenoma. They are not different names of the same thing, but names of very different application; the former is the name of a peculiar disease of the glands, spleen, &c., while the latter is a name used for any tumour that has a lymph gland-like structure. Indeed, the name lymphadenoma is now used by the highest authorities, say for cancerous disease of the skeleton, lungs, and heart, or for a malignant tumour of the kidney; so that it has become a histological rather than a pathological name, and has no *clinical* application like Hodgkin's disease. The latter is a general lymphadenoma of the glands so frequent as to require clinical recognition. This disease is generally not confined to the glands, it very commonly affects also the spleen, as well as the liver, and in both these organs consists of a development of a lymph-gland like tissue in the form of growths generally following the course of the lymphatics in the affected organs. The sections through these growths often have an angular outline and a whitish appearance that has been well compared to the appearances of pieces of suet scattered through the organ. Such are its more constant seats, but it may also be found in these cases in the walls of the intestine, especially the submucous coat, in the kidney, periosteum, dura mater, subcutaneous tissue, &c. In some cases it has one distribution, in others another, and the clinical symptoms vary somewhat in consequence, yet, in the cases where the lymphatic glands, spleen, and liver are chiefly affected, *i. e.*, in Hodgkin's disease the course of the disease is chronic and its termination accompanied by marked anæmia and dropsy, no excess of white cells being present in the blood.

It was the wide cancer-like distribution of the disease, with the absence of those so-called "cancer cells," which were formerly regarded as characteristic of malignant growths, that drew attention to this peculiar affection. The microscopic structure was first clearly recognised by Virchow as essentially like lymphatic gland structure. Subsequent writers, as Trousseau, or Cornil and Ranvier, have not added much to his description, but he unfortunately ignored the clinical importance of the disease, which they have recognised. You must notice that different examples of this disease, identified by the peculiar distribution and general appearance, show very different degrees of softness and hardness, and with these also very different clinical tendencies, the soft growing more rapidly, even becoming infectious to parts around, the harder forms growing more slowly and keeping more within their original bounds. But, soft or hard, the essential structure is very simple, and in short is that of lymphatic gland, the soft form having more cells and the hard more meshwork;

but the cells resemble lymph cells, and the meshwork is that proper to lymphatic glands, the stellate cells of the meshwork and its concentric arrangement being also quite recognisable. The very soft forms too have often large poly-nucleated cells, while the meshwork is delicate, so that they become very like soft carcinomas, only that the cells are not epithelioid but lymphoid.

The resemblance to lymph gland is expressed in the names lymphadenoma and lymphosarcoma, the latter of which is from Virchow, while the former is adopted from Cornil and Ranvier. If the new formation were limited to the enlarged glands it would never have received so much attention, but must have been regarded as a simple hypertrophy of them, with varying induration or softening; but it is most important to observe that the likeness to the structure of glands is not limited to the growth in the glands but is found in the growth in the spleen and in the liver, &c., so that the disease becomes more than a glandular hypertrophy, and it is still more curious to see that the softer examples may show a great power of contagion in their neighbourhood. Thus, in one of our cases the disease extended from the enlarged mediastinal glands across the pleura to the neighbouring lower lobe of the lung in which it spread freely. We here see a disease which begins as a simple swelling of the whole gland go on to transform other tissues around to the nature of gland in the most malignant way. This fact weighs heavily against the limitation of malignancy to local heterology of structure, for the growths are most malignant and yet homologous.

Some have thought Hodgkin's disease may be traced to irritation of the glands through local disease in the parts they draw from, say caries of the ear setting up enlargement of a cervical gland and the enlargement extending from gland to gland. A successive enlargement of the gland is often traceable, but we do not know any facts showing that it is commenced from a simple irritation.

Local lymphadenoma.—Sometimes the same disease of the glands as becomes general in Hodgkin's disease, is found limited to one gland or a few glands or a packet of glands in one region of the body. This is most frequently seen in the neck, in such a case we have known the jugular vein entirely closed by the pressure of the glands. In many such cases the glands on section do not differ, except in size, from scrofulous glands. Virchow would call such glands scrofulous, however large they may be, whenever caseous degeneration occurs in them. Yet we owe to himself the evident demonstration that caseous changes occur in cancerous syphilitic and, in short, almost every kind of tumour, so that the occurrence of the cheesy change seems an insufficient criterion. But we have seen that when enormously large these glands may evidence a scrofulous nature by setting up tuberculous pleurisy or peritonitis, which is a better proof of scrofulous nature. This occurred in an enormous cervical tumour already alluded to, which proved fatal by tuberculous pleurisy.

In other cases the tumours come so near other malignant diseases in their characters that they have formed part of the best examples of the old "encephaloid cancer." As far as we have seen, primary cancer of the lymphatic glands is always lymphadenoma, which in this malignant form may be called *lymphoid cancer*. It is very frequently seen in the cervical glands, and also, not uncommonly, in the mediastinal and bronchial, forming part of the grave clinical cases known as mediastinal cancers (part, but not all, for some of these cases are truly carcinomatous, and then the glands are affected less than the surrounding tissue). Such primary lymphoid cancer is not infrequent in the lumbar glands, forming most of the cases known as "Lobstein's retro-peritoneal cancer." Another very important seat of the occurrence of lymphoid cancer is in the glands of the mesentery, which may grow to an enormous size; we have thrice seen such cancer extend down from the glands along the lacteals and reach the attached side of the bowel, spreading then around the bowel in a circle, yet widening not narrowing it. This is the only cancer that causes expansion of the affected part of the bowel.

Scrofulous disease of the glands.—This is distinguished from hypertrophy and lymphadenoma by its tendency to undergo caseous degeneration and by its alliance with tubercle. The gland enlarges by irritative hypertrophy, so that we find increase of the lymphoid cellular structure causing at first a slightly translucent swelling, this swelling, however, has a dryness such as characterises scrofulous swelling. This dryness signifies the dense aggregation of the new cellular growth in the gland and its want of freedom, so that it compactly compresses its own vessels, thus leading to gradual loss of vitality and caseation. The caseous change shows itself by all the elements of the texture gradually losing their sharpness, outline, and wasting into fatty remains, then these fatty relics may be entirely absorbed, which is a rare and the most favorable end. More often the caseous tissue breaks down into a curdy matter which may then dry into a pasty remainder which grows calcareous, especially in the mesenteric or bronchial glands, where the stony material in long intervals afterwards may undergo enlargement by accretion until a mass of stone as great as a walnut or even much larger may be found.

But instead of thus drying up there may arise a certain degree of more acute inflammation around, uniting the diseased gland closely with surrounding parts so that they become fixed as no simple hypertrophied or lymphadenomatous glands do. This inflammation may proceed to true suppuration and the abscess open either spontaneously or by incision, leaving a deep indolent "scrofulous" ulcer.

We have, however, known these cold abscesses to recede without bursting, even when they have undermined the skin and raised it as a thin cover over the contained pus.

We must draw your attention to the dried, caseous, or purulent remains left by such indolent, scrofulous, glandular inflammation. They have just now a great pathological interest through the discoveries of Villemin, Cohnheim, and others, that such matters injected into the veins of dogs and other animals produce general tubercle, and through the application of these discoveries in Buhl's theory which supposes that their absorption from the old scar into the blood may create phthisis long after the healing of the scrofula.

Leukæmic lymphoma.—It is said by Virchow and others that in some cases leukæmia depends on enlargement of the glands, though usually on enlargement of the spleen, and that the lymphatic variety is distinguished by a smaller size of the white cells in the blood. We have not yet observed any case of leukæmia in which the spleen was not enlarged, yet the glands are sometimes enlarged with it. The structure of the glands in this disease is natural and the only distinction from simple hypertrophy is the association with leukæmia. We must mention to you that Virchow in his description of leukæmic tumours in the organs does not unite them with Hodgkin's disease, though some have quoted him to that effect. The latter disease with him is lymphosarcoma, while leukæmic tumour is lymphoma.

Tubercle.—Tubercle can be distinguished from scrofula in the glands only when it appears in its miliary form. Thus in cases of general tuberculosis you find the lymph glands often freely sprinkled with the same kind of tubercles as are found elsewhere in the body. Also in the bronchial glands corresponding to phthisical lungs, and in the mesenteric glands corresponding to phthisical intestines, you often find numerous miliary tubercles of quite characteristic appearance, as you see in this drawing. Tubercle itself, so far as it has any structure, has a coarse resemblance to the simple structure of the lymphatic glands. In large tubercles of the brain we have seen the resemblance brought pretty close by the concentric character of the meshwork we have before spoken of as characterising lymph-gland structure. Regarding tubercle as a lymphatic growth it becomes in the glands a new formation of a little mass of gland tissue, and hence would be homologous in Virchow's sense of that word. Now, Virchow regards tubercle as a heterologous and malignant lymphoma. On his view, as you know, malignancy goes with heterology, that is, with a non-resemblance to the tissue the new formation occurs in. To explain this difficulty, Virchow regards tubercle as *not commencing from the proper tissue of the gland, but arising in the septa, &c., of the gland.* We have, however, not been able to see anything to substantiate this complex view, we rather have found in tubercle of the glands a low structure of fibrils and lymphoid cells spreading through the glandular structure, and the resemblance of this low structure to gland was due to the fact that both had lymph cells and neither had very much else in which to differ from the other, excluding the vessels, &c., of the gland, which are not taken into account in Virchow's statement; in short, the intricate difficulty thus created is an altogether unnecessary refinement of a simple subject.

Relation of tubercle to scrofula.—Whether scrofulous disease with its general or widely spread large patch of caseous change is the same as tubercle with its smaller miliary grain, is a question that will be asked specially in regard of the glands, because they are the classical seat of scrofula. Leaving all theoretic and speculative grounds, there is this difference between the two conditions, that the uniform cheesy change of scrofula is never secondary to any similar change in other parts, while the miliary tubercles are secondary. This fact alone would make it convenient to keep to the separate terms now in use for the two conditions. We believe that *tubercle is the secondary form of the disease of which scrofula is the primary form.* You know that primary cancers are apt to be shapeless, and to spread out in bands and odd figures, while secondary cancer nodules are always round, for instance, those common ones in the intestines are as round as coins. We cannot easily explain this difference between primary and secondary cancers, but we believe that if the explanation were given it would apply as much to the round form of tubercle, secondary to shapeless scrofula, as to the round mass of carcinoma, secondary to the straggling lump of mediastinal cancer.

Lardaceous disease of the glands.—This is frequent when lardaceous disease is present in the abdominal viscera; the cervical glands are especially liable. The disease does not cause great swelling; the organs harden and become subpellucid; iodine brings out the characteristic walnut-wood coloured stain. Microscopic examination shows that the small arteries are, as usual, first affected; they become swollen and lustrous. The change then extends to the lymph cells, which swell and often unite into masses of glistening lardaceous matter. (For a more particular description of the change, see the description of it under Spleen.)

Cancer.—One of the first things by which a cancer in any part proves its malignancy is the infection of the corresponding lymphatic glands, and thus secondary cancer of the lymphatic glands is extremely frequent; we were going to say almost as frequent as cancer in all other parts together, but not quite so frequent as this would infer, for some cancers in vital parts kill before there is time to infect the glands; and other cancers, especially the malignant sarcomata, are remarkable for their disposition to extend to remote organs, without implicating the lymphatic glands. Perhaps it was because of this frequency that it was thought by some that cancer of the lymphatic glands was always secondary ; but you may be sure that primary cancer of the lymphatic glands does frequently occur, and then, as we just now said, has the characters of lymphoid cancer or soft lymphadenoma. We need not enumerate the kinds of cancer that appear secondarily in the glands, suffice it to say that they all do so.

Arrest of Pigments and Poisons in the Glands.—The spaces between the tissues are continuous with lymph vessels, and these lead up to the glands ; and by the way thus open, foreign substances in sufficiently finely pulverised, or rather molecular, state reach the glands. Thus some of the cinnabar and carbon used in tattooing a man's arms will be found in the axillary glands, lodged close to the afferent vessels, for the gland tissue acts as a filter, and stops the foreign substances. But this process is exhibited on the largest scale in all of us, whether we will or not, although we might object to be tattooed. The seat of it is the lungs and bronchial glands. Pigmentary matter, such as the fine dust in the air, enters the lungs ; the cilia of the trachea and bronchia waft back most of it, but some gets down to the air-cells, and penetrates the tissue. You know how the living animal membranes have recently been proved to be permeable to solid particles, thus you can understand how the dust gets into the lymph-paths. It can be traced along the course of the lymphatics, beside the arteries, in the tissue of the lung, and at last it is found accumulated at the entrance of the lymphatics into the bronchial glands. In persons who have breathed pure air for a long while, or in young persons, the amount of this pigment may be small ; but in old inhabitants of large cities, and *à fortiori*, in those engaged in very dusty occupations, such persons as coal miners, needle grinders, millstone grinders, workers in hair, tobacco, &c., you get a vast accumulation of pigment in the glands—primarily of course in the lung. Some think the pigment is principally hæmatoidin pigment, due to irritation and extravasation of blood, which subsequently undergoes changes ; but the mesenteric glands are very liable to irritation, and yet do not (except very rarely) become pigmented, so that the black matter appears, indeed, to be the dust breathed.

We should like to direct your attention to a point which has not yet received the attention it deserves ; it is the probable effect on the nutrition of the lung which this obstruction of its lymph-glands *must* have. *Must* not this be a great cause of the wasting of these organs which always accompanies pigmentary states, and especially which supervenes in old age. We have already written on the acute effects of this under Lymphatics. We believe its chronic effects are the great cause of emphysema of the lungs.

It is not only inorganic matters that are thus stopped by the glands, Animal poisons likewise are arrested by them, and this, indeed, appears to be the chief part of their function in pathology. Thus syphilitic poison rarely goes beyond the first tier of glands. If you look at this drawing which we made from the axillary gland of a lad with leprosy, whose skin was in a frightful state of disease, you will see how at the entry of each afferent duct, there is a patch of yellow leprous matter having exactly the same structure as the leprous disease which was in the skin. It had existed in the skin for seven years, and yet it had made not one tenth of an inch progress in the gland; thus the gland tenaciously prevented admixture of the poison with the blood.

DISEASES OF THE NERVOUS SYSTEM

BRAIN

Although it is convenient, in studying morbid anatomy, to take the various structures separately, it should be remembered that in the larger sense of pathology they should be considered together, since it is often impossible for one structure to be affected without another. Thus, the brain and its membranes are frequently diseased at the same time, and with them often the cranium; but it is more convenient to study these part by part. The same remarks hold good in other organs, as the lungs, where, for instance, in former times, an inflammation was meant to include all the tissues in the chest; but now, by greater refinement, we speak of pleurisy, pneumonia, and bronchitis as distinct; but in so separating diseases for the sake of study, we should remember that practically they often all exist together, and that such combinations are often more frequent than the individual affections; thus probably an acute inflammation of the chest, involving all the tissues, is as frequent as one affecting these separately, and the same also in chronic disease, as phthisis. In speaking, therefore, of the membranes and brain separately, you must remember that practically the diseases are not thus always rigidly and anatomically distinct.

Malformations.—These so usually affect the brain and all its coverings together that it will be convenient to treat of them while speaking generally of the brain as a whole.

Sometimes the lateral halves of the brain coalesce, or rather are never separated, so that the hemispheres are conjoined and the eyes develope in a united state, hence called *cyclops* or *monoculus*.

The brain or spinal cord may be absent or very imperfectly developed, *anencephalus*, *microcephalus*, &c., or the ventricles of the brain and cord, or of either alone, may be much dilated (*congenital hydrocephalus*, *congenital hydromyelus*), but the most important malformations are those wherein the head attains to a fair degree of development, but parts naturally within the skull protrude as tumours outwardly. We have already mentioned to you how often the laminæ of the spine are deficient and the meninges protrude, forming a cyst in the back; in the same manner, from an unnatural opening in the cranium, the cerebral membranes may escape and form a sac, in which may be a part of the brain itself.

The usual position for such a deficiency of bone is behind, in the occipital region, between the fontanelle and the foramen magnum, or low down in the forehead. Here, through a round opening, the cerebral contents escape. Of seventy-five examples collected by Mr Zachariah Lawrence fifty-seven were occipital and seventeen frontal, but such protrusions sometimes occur at the sides of the head, and in other cases the deficiency is about the *sella turcica*, usually just in front of it, the tumour projecting down through the roof of the mouth.

In this head, where the calvaria has been removed, you can see the hole within, and the membranes passing out to form the external sac. In this other preparation, you can better see how the cyst is formed: the arachnoid appears free, but the dura mater is lost on the external covering of integument. If the protrusion arise simply from a bag of membranes containing fluid, the affection is called *hydromeningocele*; such a case you had an opportunity of lately seeing in Mary ward. The cyst protruded from the back of the head, and was quite transparent; it spontaneously sloughed, the fluid escaped, and when the child left, the wound was healing. When the tumour contains brain structure as well as fluid the affection is simply called *hydrencephalocele*, or if there is no fluid it is termed an *encephalocele;* if the brain enters there is usually a prolongation of the ventricle within the extended portion of brain.

A very good example of hydrencephalocele is seen in this drawing of an infant, where you see hanging from the posterior part of the head a tumour almost, if not quite, as large as the head itself. In the middle of the occipital bone is a round opening, and it appears as if half the brain had been squeezed through this, so that only the anterior and part of the middle lobes of the brain were within the true cranium, which was very small, from the frontal bone being pressed down; the remaining portion was in the sac behind. The ventricles, in like manner, were divided between the two, the sac containing their hinder part, with the choroid plexuses; the posterior lobes were adherent to the membranes which formed the sac, and corresponded to the parts of the tumour on which pressure had been made from supporting the head, and which were sloughing, the corresponding portions of brain being also softened; the cerebellum was within the cranium, as usual. This shows that such a condition of head is incompatible with life, if it only be from the want of a protecting case. But in a case given by Mr Hutchinson the cerebellum was included in the tumour; and in one given by Dr Murchison there was no cerebellum at all. Minor degrees, however, of encephalocele will allow of life, so that their subjects may grow to adult age, *i. e.* if they escape surgical interference. These tumours are usually met with at the root of the nose, between and above the eyes, or more rarely in the occipital bone. When a congenital tumour is found high up in the neck, it is sometimes difficult to say whether the brain or upper spinal region is affected. In such a case, where a cyst had existed for many years, Mr Solly describes its neck or peduncle as becoming impervious—this is, it underwent a spontaneous cure—and then the sac was removed.

Malformations are probably the result of inflammatory conditions during fœtal life. Thus in this specimen you see the falx cerebri deficient at its anterior part, and the two hemispheres morbidly adherent. This, probably, was due to some inflammatory action at the time alluded to.

Deficiency of the falx you find also in connection with atrophy of the brain, thus showing it not to be a simple arrest of development of the falx but a consequence of a more general morbid action. As to the probability of the fœtal brain inflaming we have found inflammatory adhesions and effusions as early as the fourth month of fœtal life, and we shall shortly tell you of the frequency of cerebritis in the syphilitic fœtus at full term.

DURA-ARACHNOID

Hypertrophy.—In cases where the bones are thickened, as in the specimens we showed you in the first lecture, the dura mater is often much affected in the same way. This thickening has especially been found in cases of epilepsy, and in some cases of chronic mania; also, wherever the bone has been the subject of ostitis.

Atrophy.—The dura mater may often be found thinned in parts, especially along the mesian line and near the Pacchionian bodies.

Connexion of Dura-Arachnitis with Injury and Disease of the Skull.—This membrane is essentially double, its outer layer corresponds to the periosteum of bone, and belongs intimately to the bone, so that it is affected with it, and not with the organ inside; indeed, it affords a valid protection to the brain, and its outer surface may be diseased to a very great extent without this organ at all suffering. The dura mater itself is not primarily affected by great inflammatory processes, but only in connection with the skull, and thus it is not found diseased except in cases of fracture of the cranium or necrosis of the bones, &c. In the former case, it may be injured at the same time with the bone, be lacerated, and afterwards slough through the direct injury; or, at a subsequent period, after portions of cranium are removed, and the surface of the membrane exposed, it may become involved in the softening inflammatory processes. It is more rare to get suppuration about the dura mater from injury without fracture. In cases of disease of the ear and temporal bone, the contiguous dura mater may become involved, changed to a dark colour, and slough; or suppuration may rarely occur between it and the bone, lifting it off the bone in the form of an abscess. In any of these cases there is danger of implication of the sinuses, and consequent purulent infection. We have seen meningitis set up by ear disease without evident affection of the dura mater. The more chronic inflammatory processes are seen in those cases, especially in syphilis, where large portions of the calvaria have come away, and the dura mater is exposed: in these we may see the outer surface of the membrane covered with large patches of lymph, which form hard granular layers, producing much thickening of the whole membrane. It is remarkable for how long a time the inner serous surface remains unaffected in such a case. This is a very good example of a dura mater so covered with inflammatory product; it corresponds to a necrosed calvaria which we have already shown you.

In some cases, after injury to the bone the dura mater becomes adherent to the parts beneath, and if the chronic inflammatory process continue, the brain may be involved. Thus, in a man who, since receiving an injury, had suffered with epilepsy; the bone was much thickened by ostitis, as well as the dura mater beneath it, which was inseparably connected with the underlying convolutions.

Acute Inflammation of Inner Surface of Dura Mater—(Dura-arachnitis).—Recent anatomists have almost explained away the arachnoid, declaring the "parietal layer" to be an epithelium on the dura mater, and the "visceral layer" to be only the smooth outer face of the pia mater. But the space known as the arachnoid cavity, like the other great serous surfaces, offers opportunities for the formation and accumulation of morbid products; and these products arise under such special circumstances, or else are so characteristic, that it is necessary in pathology to recognise the serous nature of the arachnoid surface.

Nevertheless, what the modern anatomists have said of the relations of the arachnoid to the dura mater has a certain interesting analogy with what we shall now see, for while the anatomists say the parietal arachnoid is only the inner epithelial face of the dura mater, we, as pathologists, must tell you that the inflammations of the arachnoid occur only through the medium of injuries to the dura mater; and, again, when anatomists deny to the visceral layer any special nature, and regard the pia mater as the true and proper membrane, of which the arachnoid is a mere superficial face, we, as pathologists, must tell you that this visceral arachnoid has equally no distinct pathological existence, and *never spontaneously inflames*, nor even in spontaneous inflammation of the whole covering of the brain does it show any activity; for while the deeper vascular layer of the membrane may fill the interval between itself and the arachnoid surface with lymph, there is not any noticeable formation on the free arachnoid surface.

We find students who have no practical information on the subject suppose that, as in pericarditis, pleuritis, &c., the inflammatory products are poured out between the serous surfaces; so, in the case of the membranes of the brain, or in arachnitis as it is called, the inflammatory products are found in the arachnoid space. This is, however, wrong; in *idiopathic* inflammation, the exudation is from the pia mater beneath the visceral arachnoid, although there is some slight secretion from the other side; and thus we prefer using the more general expression, meningitis, in the idiopathic inflammation of the membranes of the brain. In true arachnitis, where the exudation is between the serous surfaces, as in pleurisy, &c., we believe the disease invariably originates from the dura mater and skull. We have never seen extensive exudation in this interarachnoid space without an injury to the head, or caries of the internal ear, or syphilitic caries or other cause of suppuration in the bones, or else through meningitis from bedsore, or from disease of the spinal column extending up to reach the brain; and thus we speak pretty confidently on the point. We have never ourselves known such an affection as this simple arachnitis spring up as a spontaneous disease, and therefore, when you meet with it we should advise you always to look for some mischief outside. When arising in this way, the interarachnoid effusion is often purulent, and so copious that it may pour out when the dura mater is removed. It may form a circumscribed abscess, limited by adhesions around; cases are recorded of successful opening and cure of such abscesses. This purulent effusion more rarely extends to the base. On this account it is called meningitis of the vertex by German pathologists; tubercular meningitis being called meningitis of the base. It is found principally on the side of the fracture or injury, being often prevented extending to the other side by the falx. If it does reach across, the amount is less there. The inner surface of the dura mater is in these cases covered with lymph and pus; with this effusion there will generally be also an exudation of lymph beneath the visceral arachnoid, as in idiopathic inflammation, forming an opaque sulphur-coloured layer in the subarachnoid space, over the convolutions; but whenever there is lymph in the arachnoid cavity you must look for a cause externally in dura mater and skull.

Although we say thus confidently that pus in the arachnoid cavity is constantly an indication of injury, or disease of the bone or dura mater, yet you will find a few examples of pus within the arachnoid sac, as an extension from the spinal canal. This has been seen by Husch in a few exceptional cases of epidemic cerebro-spinal meningitis, and Dr Murchison has given a similar case. Dura-arachnitis sometimes occurs from blows on the head without fracture.

Effusion of Blood.—When blood is met with in the interarachnoid space, it may have proceeded either from the vessels of the dura mater without or pia mater within. It rarely originates from without, since rupture of meningeal arteries through injury is attended generally by a flow of blood external to the dura mater, between that and the bone; while other causes, such as this aneurism on a meningeal artery, or any other bleeding tumours, are rare pathological curiosities. On one or two occasions, however, where, after a blow on the head, blood has been found covering the arachnoid, and no injury to the brain or dura mater could be discovered, it was considered that the source must have been some of the vessels in the dura mater, because this membrane is more immediately concerned in shocks to the bone. We have seen a few such cases; in one a boy died after a blow on the head from a stone thrown at him. There was no fracture of the skull, and no sign of injury to the brain, but much blood was in the arachnoid cavity; also a similar accident to a blind man, who fell on his head. Several such cases are recorded. These accidents generally affect elderly people through falls. The blood sometimes evidently originates in the pia mater, it may have come by spontaneous hæmorrhage, or from an injury. Thus, in fractures of the skull, if the brain have been at all bruised, and the arachnoid torn, some blood will be found in the arachnoid space, as well as under the pia mater. This is of everyday occurrence; sometimes in cases of fracture, blood is effused into the arachnoid cavity, without the dura or pia mater being evidently torn, and then some observers believe it has come from rupture of the veins that cross from the brain to the sinuses. Interarachnoid hæmorrhage arising spontaneously is not very common. We have seen it in a child, who had died of convulsions in hooping-cough; it would then be called *meningeal apoplexy;* it resembles the ecchymoses of the eye in hooping-cough; in all the other cases in which we have witnessed it (and they have not been many), the patient has had Bright's disease. In some of these it was quite unexpected, and had evidently happened only a short time before death; on removing the dura mater, a thin layer of recent black coagulum was found partly covering the hemisphere on one side; its source must have been the pia mater, though it was impossible to find the ruptured vessel.

Chronic Hæmorrhagic Dura-arachnitis.—Pachymeningitis.—Hæmatoma.—Changes connected with old hæmorrhage.—We are now upon much debated ground, namely, that which concerns the presence of certain layers of false membrane with more or less altered blood in them, attached to the dura mater, sometimes so closely as to thicken it (hence called *pachymeningitis*); sometimes with effusions of recent blood between the layers of false membrane, so as to make a blood tumour (hence called *hæmatoma of the dura mater*). Blood, recent or old, coexists in them with false membrane, such as we see in serous inflammations, and the question is whether the blood causes the false membrane by inflammation, or the false membrane, which is vascular, bleeds, producing the blood. We will first take one certain step, which cases that come under our own observation place beyond a doubt, proving that effused blood will produce these layers. We have seen several cases where the prominent lower parts of the middle and anterior lobes of the brain, showed old bruising so exactly in the manner constantly seen in cases of severe injury to the head, that there could be no doubt such an injury formerly had been suffered. This was, in two of the cases rendered certain by the presence of an old-healed depressed fracture of the left parietal bone. Now, in these cases, a considerable extent of the dura mater, reaching sometimes to the vertex, had a lining consisting of an adherent yet separate layer, identical with the so-called pachymeningitis; that is, a brick-dust coloured layer, which, under the microscope, showed a quite beautiful structure—the characteristic structure of pachymeningitis. It was full of wide capillary meshwork of long tortuous vessels, which in many parts showed varicose dilatations; supporting the meshes was a delicate texture of spindle-cell connective tissue, in which were numerous crystals and glomerules of hæmatoidin, which gave the red colour to the membrane. These membranes were long ago described accurately by Wedl, without their connection with hæmorrhage being suspected. The examples just given will show that an old traumatic effusion of blood *will cause* the false membranes in question; and we must therefore believe that such membranes are so caused, until it is equally clearly shown that they can arise by spontaneous inflammation. The other mode of origin for these membranes advanced by Virchow, and generally accepted abroad, is this: the inner surface of the dura mater is thought to produce by irritation a layer of inflammatory false membrane, which becomes highly vascular. As stated by Rindfleisch, this view goes on to say that new layers form on the surface, and are again highly vascular, while the old layers beneath contract on and obstruct the returning circulation through the vessels of the superficial layer, so that these burst, causing effusion between the layers, leading thus to larger or smaller effusions of blood. The larger effusions may distend the false membrane into the form of a cyst, but the smaller will be found long after as old altered blood-pigment; some go so far as to trace the share of the epithelium in the production of the original false membrane, which is said to be evidently considerable, as its cells are swollen much around the edge of the new formation. But long ago Mr Prescott Hewett had traced the whole process in many cases to an effusion of blood, and shown how this, in course of time, assumes the form and structure of a serous cyst; and there is no doubt that if, from injury, blood be effused between the arachnoid surfaces, it forms a thin layer over one or the other hemisphere, and coagulates; soon the fluid part is absorbed, and the fibrinous layer undergoes organization, while the coloring matter passes through the usual changes; the colour alters to a brown hue, the corpuscles are broken up, and the hæmatin crystallises. Perhaps, however, both methods of formation occur in different cases. Yet we think that hæmorrhage is generally the first step in the process, though probably bleeding into the new membrane happens afterwards, as described by Virchow. In a case we examined not long ago, where death occurred three weeks after the injury, a thin brown layer covered the hemispheres and inside of the dura mater in this manner. The crystals of which we speak are named *hæmatoidin*, and are formed from the hæmatine; they are of a deep ruby-red colour, and in shape oblique rhombic prisms, sometimes a little more irregular, and if seen for the first time you might perhaps mistake them for uric acid. It might be important to know at what time such crystallization takes place, so as to ascertain the duration of the effusion, but of this we cannot speak more positively than that they may be made in a short time artificially, but in the human body we never saw them within three weeks after a sanguineous effusion; in recently poured-out blood the hæmatine is in irregular granular masses. It is after a longer period, as several years, that such a film of blood will form a distinct vascular membrane, such as we have described.

When the effusion of blood is large, and the inner and outer surfaces of the clot thus widely separated for a long time, a distinct layer forms on each face of it, which may remain permanently separate, so as to produce a cyst, resembling the old apoplectic cysts, within the brain, which we shall soon describe.

These cysts may thicken enormously. Thus, in one case we met with in an idiot boy some time ago, the cyst which covered the whole upper surface of one hemisphere was nearly as thick as the dura mater, to which it adhered more firmly than to the arachnoid. Its contents consisted of a whitish paste, glistening with cholesterine. Some such cysts have been met with containing no sign or little signs of blood, but containing a watery fluid. They have been called by Virchow *hygroma*.

Syphilitic Inflammation of the Dura Mater.—This is often met with; the outer surface of the membrane and the bone are generally affected; at the same time the disease tends strongly to extend to the inner surface, and cause adhesion to the brain at the affected spot, or it may excite suppurative arachnitis. The disease produces a fleshy swelling, pink or red, from congestion and vascularity of the new products, and early showing a caseous patch in the centre of the affected portion. Of its occasional resemblance to sarcoma we shall presently speak.

Adhesions.—In children and old people the dura mater is naturally adherent to the skull. In young infants you have often, no doubt, found great difficulty in separating the two, and in old people you have been obliged to use considerable force to remove the calvaria along the median line and over the frontal bone; and even then the membrane has been most likely torn, and portions remained adherent to the interior of the skull. You should expect to find these adhesions in the young and old, but in the adult they are unnatural, and show disease. Thus you may meet with them in all cases where the bones are thickened, as in the instances before mentioned, but especially in those cases where a distinct ostitis has resulted from a blow; in any such examples the membrane may be adherent, and in exceptional instances, where lymph has been formed on the inner side of the dura mater, it may be found also closely connected with the brain.

Separation of the dura mater from the cranium usually arises from effusion of blood, owing to laceration of a meningeal artery, usually the middle, in cases of fracture of the skull, perhaps more often in old people whose meningeal arteries run in deeper grooves of the bone; sometimes, also from laceration of its great sinuses. Blood is poured out, between the two, forming a tumour, which presses on the brain, produces compression of that organ. In this case there was no other bleeding within the skull; you will notice that the clot in such cases is curiously firm and hard, and of a dull purple colour. This is due to the compression of it between the bone and dura mater, which causes absorption of the more liquid parts. It is to these cases that trephining is especially applicable, such clots will generally be found about the large branches of the middle meningeal artery. It is not often that they exceed an ounce, while the clot of fatal apoplexy is generally three to four ounces; but there is usually a good deal of blood effused inside in the arachnoid and subarachnoid spaces at the same time, hence the compression.

The dura arachnoid is sometimes lacerated in injuries, so that the brain protrudes forming a *hernia cerebri*. Often, however, it is after some days' inflammatory action in and beneath the dura mater that the perforation occurs spontaneously or by operation. We have sometimes seen a laceration of the longitudinal or lateral sinus causing fatal hæmorrhage in fracture of the skull. For example, we will show you at the top of the lambdoidal suture in this skull a Wormian bone so turned round and its sharp point turned in, that it was thrust into the longitudinal sinus, yet the little bone is so fast set in its new position that you cannot pull it out. Sometimes a simple fissure along the skull tears open the sinus and then fatal bleeding may occur if there is a free external wound.

Morbid Growths.—Among the most frequent changes in the dura-arachnoid is a production of *bone*. This may proceed until the whole structure is changed into a bony case, and appears like one calvaria within another. In this dura mater you see large patches of bone in different places, whereby nearly the whole of it is ossified, yet there were no brain symptoms. In lesser degrees it is very commonly met with, and especially in the falx major, which is its favourite seat. In this specimen, from a woman with melancholia, you see large pieces and plates of bone on each side of the falx; and in one case the other day, you remember we found a piece like a tooth. This we carefully examined by the microscope, and found, as in all other similar specimens, that the structure was true bone, the lacunæ and canaliculi being admirably displayed. Several such specimens are put up and preserved in our microscopic drawers. You may recollect that we told you that all the so-called ossifications of the heart and blood-vessels consisted merely of very simple earthy concretions, so that they should rather be called cretifications or petrifactions; whereas now you may remember that all the ossifications of the membranes of the brain and spinal cord consist of true bone.

In speaking of cancer of the skull, we stated that in many instances, it appeared to commence in the dura mater, and then penetrate the bone. Its existence may not be suspected until an attempt is made to strip the dura mater from the skull, when the cancerous deposit is opened; it is then seen to form round soft patches on the membrane, and on the corresponding surface of the skull the bone is seen eaten away in its internal table, and is occupied by the same cancerous material. Although most authorities agree in describing these multiple round tumours on the outside of the dura mater as sometimes belonging to one and sometimes to the other, yet we think these tumours more properly belong to the bone, as you will see by observing first how frequently they coexist with general cancer of the skeleton, and second how close a resemblance there is between all the cases. For a notice of their very variable structure see p. 62. Sometimes single tumours project inwardly towards the brain, but they may be found in the substance of the membrane implicating the bone, and also pressing inwards; and in such cases again a question may arise whether the bone was not first involved. The growths that spring from the inner surface of the dura mater, as they rise towards the interior of the skull, take within it the form of low protuberances; one of these may swell at the most prominent part so as to acquire a constricted appearance at the attachment, and we have found such a polypoid tumour growing across the arachnoid cavity into the substance of the brain to some depth. Such projecting tumours vary from a fibrous to a fleshy consistence. Their microscopic structure is commonly that of well-characterised *sarcoma* or *fibro-sarcoma* (Plate II), the consistence being more firm the more fibrous the structure; some examples have a mucous intercellular substance, and hence would be called myxoma (Plate III). These, however, have generally affected the dura arachnoid of the spinal cord. An interesting form of tumour is described by Virchow under the name Psammoma, these, he says, affect the parietal dura mater principally as hemispherical excrescences separable without difficulty; they are tolerably firm, of reddish or marrowy white colour, and on section show a quantity of sand-like grains, which under water fall out or remain connected with the loose vascular meshwork of which the substance is composed. He connects the sandy nature of these tumours with the sand grains found naturally on the dura arachnoid about the sella turcica and elsewhere.

All these tumours show a predilection for the neighbourhood of the body of the sphenoid and next for the falx at its anterior part. As a rule, internally projecting tumours come more from the middle region of base of the skull than any other part of it. This leads us to remark that such tumours are often difficult to distinguish from syphilitic growths, which also are mostly found about the sphenoid, because these too are liable to undergo a fatty or caseous degeneration that you not infrequently meet with in sarcomas; sometimes it is only by a careful consideration of the attendant conditions that you can tell syphilitic gumma of the dura mater from sarcoma.

The dura mater is, as you know, smooth on its inner face, being lined by the arachnoid, which, however, here consists of little more than a layer of epithelium. In considering growths from the inner surface the question comes, do they arise from the dura mater or from the arachnoid? This question acquires great interest with respect to certain examples of epithelial cancer, which have been met with generally in the occipital region. But such a question is very difficult to answer, epithelial cancers usually belong to surfaces where a thick coating of epithelium is found, as the skin or mouth, &c. The arachnoid is, we believe, the only serous surface which has been known to give rise to epithelial cancer; no one has yet shown what share the epithelium has in it. Here is a small villous growth from the dura mater over the basilar process. The inner surface of the dura mater, like the outer surface of the pia mater, produces Pacchionian bodies. For the development of these see p. 206.

Hydatids, which are occasionally found in the brain, may also develop within the arachnoid cavity, as in one case which occurred some years ago at the Surrey Dispensary. On removing the dura mater, two or three cysticerci fell off from the surface, being loose, or only slightly connected with the arachnoid; they were of moderate size. This is an old museum preparation, showing a cysticercus from the surface.

PIA-ARACHNOID AND SUBARACHNOID SPACE

Congestion.—We must warn the inexperienced against mistaking a mere fulness of the blood-vessels, arising from accidental causes, for a morbid congestion; and even against regarding the latter, which is often only of trivial importance, for an active state. Those who are not much acquainted with post-mortem appearances, are too apt to regard much blood in the brain as a pathological condition, and this is a state very likely to be present in those cases requiring examination through an order from the coroner. For in the first place several days may have elapsed since death, and again, perhaps, the head only may have been opened; besides, if death has been sudden, which is very likely to have been the case, there is yet another reason for a fulness of blood, for there is more blood in the body of a person struck down in comparative health, so that if death be sudden, there will be an excess of it stagnating in the organs, which excess from the body having been supine for some days, the brain has more than its share. Further, it is very important to remember that if the head be examined first, the vessels are necessarily more full than if the body be previously opened, for in the latter case, the veins being cut and jugulars emptied, the blood flows out from the cerebral sinuses and surface. You may observe this difference constantly in the post-mortem room, according as the body or head is first examined. But if these accidents do not intrude, so that the head is opened after the body, and the person only dead a short time, even then if the vessels of the dura mater, pia mater, and brain itself be very full of blood, all this is generally unimportant, and points merely to the cause of death by asphyxia. For in all pulmonary obstructions, whether primary or secondary, the brain is found congested like all the other organs.

There is no doubt that congestion of the brain is a real cause of serious symptoms and even of death. We particularly ask you not to suppose that we are denying the existence of congestion of the brain as a fatal disease, what we wish to say is that a fulness of the vessels after death cannot *prove* the occurrence of the primary congestion, and in fact *post-mortem fulness of the vessels of the brain and its membranes is of no pathological value whatever* as showing brain disease, because asphyxia produces it. On the other hand anæmia of the brain does exclude congestion and also proves that death was not through asphyxia, which always produces congestion. Our reason for saying all this so confidently, when even now you find writers in systems of medicine speaking of redness of the cerebral membranes as the great sign of inflammation, &c., is that we have seen all degrees of intensity of such congestion when there was no trace of cerebral disturbance during the life of the individual, and this we have seen not occasionally, but as a common occurrence. Again, it is not only the experience of morbid anatomists which declares intracranial congestion to be thus valueless, we will quote for you a passage from Kussmaul and Tenner's account of their elaborate researches on the effects of hæmorrhage as causing epilepsy, they say, "We could never deduce any results from the post-mortem examinations undertaken with a view to determine the state of fulness before death of the most important parts of the vascular system, viz. of the arteries and arterial capillaries, and even in the most favorable instance, when similar inquiries were directed towards the veins, our results could only be looked upon as approximate."

Thus, you will see that it is only for lack of information that any one would speak of congestion of the brain as a proof of a cause of death. You may *infer* reasonably from the circumstances of particular cases that death occurred from congestion of the brain, but you cannot *prove* it after death by inspection. We shall presently endeavour to show you in what degree certain appearances of the brain are evidences of congestion.

Air in the Subarachnoid Space.—We speak of this because it is often met with, and students are constantly asking questions about it; and, moreover, we have seen it mentioned in a medical journal as a pathological condition. It arises simply from air rushing beneath the Arachnoid when the calvaria is removed. You may not meet with it yourselves so often as you see it here, where the opening of the head is more speedily effected than one usually sees in private. You will observe, that in sawing around the head, the visceral arachnoid is slightly wounded, and then, when the lever is inserted under the bone, and the calvaria is suddenly pulled off, a vacuum is produced, and air rushes through the opening into the space beneath, and traverses the course of the blood-vessels, and thus arise the bubbles of air which have attracted the notice of some of you.

Œdema and Subarachnoid Effusion.—Nothing is more common than to see the subarachnoid fluid increased in quantity, indeed, being the first thing observed on opening the skull, it often attracts much attention and much more than it deserves; for as a rule, it denotes merely a shrunken or wasted state of the brain accompanying a similar state of the other organs of the body, so that it is very common on our post-mortem table. In persons long ill with chronic wasting diseases, the brain shrinks, the convolutions lose their plumpness and separate. The visceral arachnoid still maintains its close apposition to the parietal; but the pia-mater, fastened to the brain by its close vascular connexion with it, follows the brain so that thus the subarachnoid space between the brain and arachnoid enlarges and is filled with fluid. This you see bagging down in various places when the dura mater is removed, while the brain itself is generally pale. If the exudation were inflammatory it would be plastic or purulent, accompanied by congestion and other characters. Neither does the œdema accompany a dropsy of other parts, as you might imagine, for there is, indeed, no room for dropsical effusion unless the brain wastes; but, of course, when dropsy occurs elsewhere, there may be an increased wateriness of the brain and its membranes. It is important to know that this subarachnoid effusion is a mere passive condition, and denotes only simple atrophy and not an inflammatory process, for it often happens that you wish to know, by post-mortem examination, whether certain symptoms really arose from a diseased condition of the brain or not. Thus in a case of advanced tuberculosis or phthisis, the patient has towards its termination, delirium, or other brain symptoms, and you may suspect some disease in the brain similar to that in other parts; but upon examination you find the brain shrunken, with fluid on the surface, a condition denoting mere atrophy, thus showing there was no inflammation, for any active tubercular condition would be accompanied by effusion in the ventricles, and the brain would be swelled out and flattened against the skull, the convolutions being pressed together, so that there might be no fluid at all apparent.

We thus do not agree with authors who, like Rindfleisch, would trace an external hydrocephalus to irritation of the brain, saying that the brain is compressed by overfilled vessels and so atrophied. Indeed, the complex hypothesis used in support of that view is both unnecessary and incongruous with the facts observed. It is *incongruous*, firstly, because the fluid is not a serum or highly albuminous fluid, such as is always the result of dropsies or irritative effusions, but is almost free from albumen, and, in short, is identical with the natural subarachnoid fluid; and, secondly, because the brain is pale in these cases, as we have before said; whereas if the effusion were active, the brain would be expected to show congestion rather than paleness; and, thirdly, because we constantly see this effusion in the bodies of persons who have had no morbid irritation of the brain, unless the ordinary course of life can be described as morbid irritation, you will find that the presence of the fluid and the coexistent wasting of the brain increases with age, and with the degree of marasmus a chronic disease has caused; fourthly, the chronic thickening of the membranes which always accompanies the effusion cannot be set down to irritation, because it lies always along the line of the longitudinal sinus, as we shall presently see. Again, it is *unnecessary*, because a simple wasting of the brain entirely explains the excess of the subarachnoid fluid on its surface. For this fluid is, indeed, naturally designed as a compensating supply for the purpose of filling up the skull when the brain falls in volume in physiological disuse, or through other causes of diminished blood pressure. So that if we do not deny that the brain wastes at all, and if we allow that though in a less degree it wastes like other organs in wasting disease, then we see that there *must* be an excess of subarachnoid fluid to compensate the cranial cavity. It is true that you find this wasting in drunkards and sufferers from mental exhaustion in the middle periods of life; but we shall give you reasons for believing that in such cases the brain is simply wasted. Some of you may be thinking that if this superficial fluid were in great quantity it would amount to a disease, and constitute *external hydrocephalus.* We may therefore at once say that we are unacquainted with any such disease. We believe there are some who consider such an affection does exist, but we have very strong doubts about it. In the case of the lad Cardinal, whose immense skull we the other day showed you, it has been thought by some that the effusion was external to the brain, because when the cranium was opened, several pints of fluid poured out, and the brain was found at the base; but on reading the account, we think it is very clear that the fluid had during his lifetime been contained in the immensely distended ventricles, and that the brain was unrolled and occupied the circumference of the skull, but that just before his death the fluid burst through the corpus callosum, and thus the peculiar condition met with.

Chronic Thickening of the Pia-Arachnoid; Pacchionian bodies, &c.— We will mention these immediately after the account of excess of subarachnoid fluid we have just given, because all these conditions constantly accompany each other and evidently are closely connected. The chronic thickenings we are now considering always strike the inexperienced very forcibly, and often we have found it a great surprise to beginners that the thick, white, nodulated band along the course of the longitudinal fissure is not more spoken of at post-mortem examinations. Let us consider first the anatomy, and then shortly the signification of this thickening.

In a typical case you find the proper membranes of an old person's brain to show a whitish opacity over the vertex, but you will always see that the white opacity is greatest on the border of the longitudinal fissure for about an inch breadth, then it diminishes outwards, so that as you turn to the lateral aspect of the brain, the membranes are not remarkably thick, and at the base over the convolutions they are quite as thin as in early life. In an extreme example you see the thickening come down along the Sylvian fissure, and you also see patches on the posterior lobe corresponding to the points of entrance of the veins of the lower part of the cerebrum into the lateral sinus. Another point where you will not fail to find the thickening is around the spot of exit of the venæ Galeni. Again, in some cases you see the membrane over the diamond-shaped space at the base of the brain thicker than natural; but when there is very marked thickening of this part of the membrane, our experience leads us to expect some former positive inflammation to appear in the history. Nevertheless it is common enough to find a slight thickening of the membranes at this spot, continuous with the thickening down the Sylvian fissure in the passive and senile states we are now speaking of.

Next you must notice that on this line of thickened membrane the so-called *Pacchionian bodies* are more numerous than usual, forming little white villous masses. Look carefully at their position, and notice that they show an unquestionable relation to the points of exit of the cerebral veins that join the longitudinal sinus, being most developed around the more posterior of these veins, which, you will remember, slope more forwards in their course to join the sinus. In extreme cases you may find a tuft or two of these bodies where the small veins from the posterior lobe join the petrosal and lateral sinuses; if you notice them carefully you will find they arise from the pia-arachnoid membrane at spots where its pia and arachnoid surfaces are blended. They are composed of a delicate fibrous tissue covered with epithelium, and are devoid of blood-vessels. At their origin they come in contact with the dura arachnoid, which there bears a few of them on its surface also. They grow, soft as they are, through the tough dura mater, and penetrate, perhaps, into the longitudinal sinuses, and extending through the dura mater, they reach and excavate the skull, even pushing their way through this, so that they would perforate it; but usually the outer bony lamella rises a little above them as in this specimen. Yet they are very soft; it is like the penetration of tree roots into apparently solid stone, the searching half-fluid pressure finding pores which the growth enters and dilates. Such are the Pacchionian bodies. They appear soon after birth and go on steadily increasing until, in old age, they reach a considerable size. They are sometimes found in a state of fatty degeneration or calcified, having in them sandy grains of "brainsand," so that they may be termed "psammoma." The sand grains are little balls of lime salts, separately encysted in fibre. We wish you to notice that there is a close relation between the development of Pacchionian bodies and the thickening of the membranes we have described. Again, there is a close relation between this same thickening and the quantity of subarachnoid fluid, so that when the membranes are thick the quantity of fluid is large, and *vice versâ*. Also notice that two other conditions accompany these changes. The bone thickens inwards, deepening the meningeal grooves, and the brain grows firmer while it wastes. This firmness of the brain, with the thickness and toughness of the membranes, renders it very easy to strip the membranes from the brain in elderly subjects. For the thick opaque membrane bears a hard pull without tearing, and the brain is separated from the surface already by the wider subarachnoid space. The reverse of the condition in young people makes separation of the membranes difficult in them.

So much for the anatomical appearances of these changes; now let us consider them in the endeavour to explain them. You must first notice that the cerebral circulation is conducted under certain peculiar difficulties. Indeed, nature places certain difficulties in the way of a free circulation through the brain, which will not have escaped your thoughtful anatomical reflections. We especially allude to the course of the upper cerebral veins, which is directed forwards, while the longitudinal sinus they enter runs backwards; the entering stream thus opposing the current it joins. Without attempting to give a detailed explanation of this, we see that, if the stream is feeble, this impediment to the exit of venous blood will prevent the brain becoming too easily emptied by gravitation in the erect posture, which would result in faintness. This opposition of the venous currents is, indeed, we believe, one of the most important means of accommodation of our species to the erect posture, though we do not think it has yet received any notice in that light. But while there is thus an advantage during feebler states of the blood-stream, the impediment caused by the opposition of current is a serious source of congestion in states of active pressure of circulation, so that the hinder cerebral veins, which enter most obliquely, and have the greatest backward rush in the sinus to resist, are greatly impeded in the discharge of their blood, and are thus held in a state of congestion. Now, you know that the stream is strongest in the centre of a vessel, and weakest at its side, and hence you will see that the lateral part of the obstructed stream will feel the impediment most seriously; but this lateral part is that which receives the last venules from the membrane around the exit, and thus it comes about that the effect of the stasis induced by the obliquity of the veins is found to accumulate around the exit of the veins. The effects of such congestion we are familiar with in heart disease, &c.; they are summed up thus:—An overgrowth of fibrous tissue, and a loss of the higher functioning elements, also a constriction of blood-vessels by the excess of fibre. These are accordingly the results we see, so that in the neighbourhood of the exit of the veins you get a gradually great thickening of the fibre of the membranes of the brain; while very close up about the exit, where the congestion was at its extreme, you meet with papillose outgrowths from the arachnoid, such as we occasionally see on the surface of the liver in cases of very protracted and severe cardiac congestion. These papillose outgrowths are the *Pacchionian bodies.*

The same chronic thickening of the connective substance, with wasting of the functioning part of the organ, similar to what occurs in the liver or old cardiac obstructions, will explain how it is the brain grows harder and firmer. It undergoes, indeed, a slight general sclerosis through the impediment we are describing. The wasting of the brain induces a complementary excess of subarachnoid fluid, whose part it is to fill the space from which the brain recedes; it appears also that the shrinking brain keeps a sort of suction upon the dura mater, tending to fill its vessels, and so induce thickening of the inner surface of the skull, but whether this be the means or no, there is constantly an inward thickening of the skull, serving to reduce its cavity in some proportion to the decrease of the brain.

Meningitis, or Inflammation of the Pia Arachnoid.—It is in connection with the pia arachnoid and the subarachnoid space that the products are formed in inflammation of the membranes of the brain, and thus it is under this head that some of the most important acute diseases of the brain are to be noticed. Dura arachnitis, with its effusion between the arachnoid surfaces, is an affection, as we told you, intimately connected with disease of dura mater, and arises from some external cause; whilst the effusion beneath the arachnoid, and immediately in connection with the cerebral substance, has its cause within, and constitutes the idiopathic inflammation of the membranes; with this, however, there is constantly a very slight exudation on the free serous surface; but this is insignificant compared with the purulent arachnitis in the same part, arising from injury, &c., of which we before spoke. In ordinary idiopathic meningitis, the effusion, be it lymph or pus, is poured out from the pia mater; a structure which, as you know, consists of a most rich plexus of vessels, held together by delicate areolar tissue, following all the inequalities of the convolutions, and lying beneath the level surface of the visceral arachnoid membrane, with which it is connected by fine filaments, forming a delicate texture that crosses the subarachnoid space. When the pia mater is affected there can be no doubt that the adjacent brain structure is also involved, and the ventricles also suffer, for into them the choroid plexus of the pia mater projects, carrying with it the inflammation; as, however, the changes are less appreciable here than in the membranes, we are accustomed to speak of the affection as meningitis, although, perhaps, in many cases the whole cerebral structures are involved.

Acute meningitis or, more strictly speaking, pia-arachnitis, is mainly of two kinds, *simple* and *tubercular*, the latter being also known as acute hydrocephalus. It is highly important to distinguish between these two forms, the peculiarities of each not being so generally insisted on as they should be; but they are very characteristic.

Simple Meningitis—Pia-Arachnitis.—In this form of disease you find the surface of the brain, in the space beneath the visceral arachnoid, covered with lymph or purulent lymph of a greenish colour. This is especially seen in the sulci between the convolutions, but may exist in such great quantity, that the cineritious structure is quite hidden; and thus, directly you remove the dura mater, you are struck with seeing this inflammatory effusion completely covering both hemispheres, as if a layer of green purulent lymph had been poured over them: this appearance you see in these drawings. On placing the finger on the brain, you perceive at once that the effusion is beneath the arachnoid, and cannot be removed without cutting this membrane. On scraping the external surface, it is likely that a small quantity of exudation may be seen on the scalpel, for it is scarcely possible for such an acute inflammation to exist on one side of a membrane without the other being slightly affected, yet on microscopic examination of the matters removed by scraping, we have often found only epithelium and detritus. On removing the brain you will find the sides covered in the same manner, but to a less degree; and on looking at the base, there is generally but little effusion there. The ventricles are not necessarily, we think, affected; but generally the fluid in them is increased, and is somewhat turbid. On attempting to remove the pia mater from the surface of the convolutions, the cineritious structure is found to stick to it, and to tear when any force is used, showing that it has also been involved; and, indeed, it would be impossible to say in such cases what parts of the brain have not suffered, though it is only on the free surfaces that the changes are markedly perceptible. In one of the last cases of this kind which we examined, the sinuses of the dura mater were filled with firm coagula. You must remember that no tubercles are found in this disease, either in the brain or any other part of the body, indeed, its pathology is scarcely known, though, in all probability, it occurs in the exanthemata; for example, as a sequence of scarlatina. It is a very acute disease, running its course in three or four days. Not many months ago, we examined a child with this form of meningitis, who had been ill only three days; every part of the body except the brain was healthy. It is possible it may originate in injury, in the same way as the dura-arachnitis of which we spoke; but we have no proof that this has ever been the case. We have seen two or three instances where an inflammation of the spinal cord has extended up to the brain, and produced a meningitis, with the greatest amount of lymph at the base. These cases have closely resembled the epidemic cerebro-spinal meningitis.

Tubercular Meningitis, or Acute Hydrocephalus.—This is a form of disease quite peculiar, and easy to recognise; for, apart from the presence of tubercles, the characters of the inflammation are such that you may know at once what is the kind of affection you have before you. The disease is less acute than that last mentioned, generally running a course of three or four weeks, with symptoms less violent. On removing the dura mater, you see the surface of the brain flattened, owing to the pressure which has been exerted by the ventricular fluid within forcing the hemispheres against the skull. The convolutions are thus pressed together, the sulci almost obliterated, so that the brain may look like an even mass, with vessels ramifying over it. The arachnoid will have somewhat lost its lustre, and on scraping it very little indeed can be obtained on the scalpel. Beneath the arachnoid no lymph is seen, unless perhaps, on looking at the sides, a small quantity may be observed in one or two sulci; thus the surface of the brain presents no marked inflammatory products, as in the acute and simple form of arachnitis just described. On removing the brain, very distinctive appearances are seen, chiefly in the presence of a little tenacious lymph at the base, in the diamond-shaped space, and about the pons Varolii, and cerebellum. The collection of lymph in these parts is so characteristic, that German pathologists call this disease "meningitis of the base." If this lymph be examined, some tubercles may be found in it attached to the small vessels that cross the space. Similar exudation is also generally seen proceeding upwards in the fissure of Sylvius, so that, if the lobes be separated, a quantity of it may be found connecting them together. The true miliary tubercles are not generally seen from the surface of the brain, but must be searched for in the pia mater after its separation. They are readily brought to view by stripping off the visceral arachnoid, together with the pia mater, and carefully examining the portions of the latter membrane which dip down between the convolutions, it is on these especially that you will perceive the tubercles. It is best to place the membrane in water over a dark surface, when the tubercles appear as white dots, such as you can see in this preparation. Probably tubercles sometimes exist before the inflammatory exudation occurs, but during the inflammation a still further and more rapid production of them takes place, in the same way as in the phthisical lung and other tuberculous organs. We are under the impression also that the simple miliary tubercles are not of long duration before they show signs of their presence, we think this because in many children who have died of general tuberculosis, there have been no cerebral symptoms; and when we have carefully examined the membranes to see if, perchance, any tubercles might be present, we have hitherto always failed to find them; but we have met with some facts which prove that the tubercles *may* be produced before the inflammation. Thus, we have found some of the tubercles so large, and so altered by cheesy changes, that they must have been older than the inflammation of two weeks' duration; and we once had a case of phthisis where a man had been quite sensible to the time of his death, and yet in the membranes of the brain were many conspicuous tubercles, some of them large and caseous, like those we have just now alluded to. It thus appears that sometimes coarse miliary tubercles precede the general tubercular inflammation, in which the crowds of minute tubercle appear, though this circumstance is only occasional. And now as regards the ventricles. On slicing the brain until they are reached, you find the roof bulging out, and the corpus callosum lifted and carried backwards, owing to the quantity of fluid present, which reaches sometimes to as much as four ounces, though it is generally less; the fluid is itself more opaque than in health, and becomes somewhat more so on boiling, and we believe the specific gravity is increased, this having been 1010 in some cases which we have examined. The circumference of the ventricle is soft, sometimes so exceedingly soft as to be quite diffluent; and this is more the case in the central parts than at the sides: thus the fornix, septum lucidum, &c., are sometimes quite broken down, shreds of the tissue hanging into the ventricles. If the septum be not broken, the foramen of Monro between the two cavities is found enlarged. Much controversy formerly existed as to the cause of this softening, whether it be inflammatory, or due merely to the ventricular effusion; but there is no doubt it is an inflammatory œdema, for the microscope reveals inflammatory products, and a mere soddening of the cerebral tissue will fail to produce a like effect. The microscope does not show, it is true, those granule masses so characteristic of subinflammatory states, but it shows the elements of these, or the fatty granules collected along the course of the capillary vessels, to explain which a passive softening is not sufficient. If the surface of the ventricles be carefully examined, it will sometimes be found granular. This may be overlooked, unless the brain be held to the light, but then the surface appears as if it had had some fine sand sprinkled over it; this condition may continue downwards into the fourth ventricle. As we said, when speaking of simple meningitis, we are apt to regard these affections as inflammation of the membranes only; but, in all probability, the brain structure is more or less involved, though the evidence of its disease is less appreciable. The superficial part of the brain can hardly avoid suffering when the mater is affected; but its implication is demonstrated, even to the n eye, by the softness and adhesiveness of the cineritious structure. Thus, in attempting to remove the membranes, the grey matter comes off with them and adheres to them, and tears up in its layers, which may be thus separated from one another.

The microscope will make quite clear to you the degree and manner in which the brain is implicated. If in the first place, you strip a little piece of the membrane from a convolution in the Sylvian fissure and examine it by a low power you will see that the tubercles follow the course of the small arteries. If then you choose a favorable spot, and apply a higher power you will see that the tubercles which are composed of well-formed lymphoid corpuscles are enclosed between the vessel and its perivascular sheath, often compressing the vessel and narrowing it. If now you harden a portion of the brain with the membrane attached, and cut sections through both brain and membrane you see that the tubercles, still within the sheath, follow the vessels, as they enter into the cortex of the brain. Thus one understands how the functions of the brain should show such severe derangement in tubercular meningitis. In no other part of the body do the tubercles so clearly limit themselves to the course of the blood vessels. This relation has given rise to several hypotheses; some observers think there is a multiplication of cells that form an endothelium to the perivascular sheath, others prefer a view that supposes the corpuscles of the outer coat of the small artery to multiply. Others yet affect a theory of outwandering of white cells of the blood into the sheath. We have examined on this point very carefully, and have seen that the tubercles certainly are close around the small arteries; but as to the hypotheses mentioned, the difficulty of seeing the actual truth by direct observation is so great when it is contrasted with the ease with which a hypothesis is made, that it is no wonder hypothesis takes the place of observation in the views of those observers who have studied this question.

To recapitulate: the distinctive characters of tubercular meningitis are, the absence of lymph on the surface of the brain (in this it differs from simple meningitis), a flattening of the hemispheres, lymph at the base, tubercles in the pia mater following the course of the vessels, and increased fluid in the ventricles, with softening of their walls. Even if tubercles were not visible to the naked eye, the rest of these characters would at once indicate the nature of the disease, and the microscope will always find the tubercles along the vessels.

Syphilitic Meningitis.—We have occasionally though rarely met with a primary pia-arachnitis in cases of decided constitutional syphilis. These cases resembled tubercular meningitis more than the acute simple form. But there was more lymph effused, and the tubercle granulations were absent. In neither of the cases was there a syphilitic affection of the dura mater. The disease was acute. Lancereaux mentions a few such cases, and we are convinced that syphilis is an occasional cause of acute meningitis.

Hæmorrhage beneath the Arachnoid.—This occurs under the same circumstances as bleeding between the arachnoid membranes already mentioned. The blood may run beneath the arachnoid when the brain is injured, or when meningeal apoplexy is a spontaneous disease, or when an apoplexy reaches the surface, or an aneurism bursts, or a tumour bleeds there. Sometimes meningeal apoplexy occurs as a fatal result of purpura or scurvy. It is not uncommon, too, to find blood beneath the arachnoid, at the base of the brain, when blood bursts into the lateral ventricles, and then passes by the third to the fourth ventricle, and from thence into the subarachnoid space of the medulla oblongata, pons Varolii, and adjacent parts. As a spontaneous apoplexy it is said to be almost peculiar to advanced life, but we have met with it in a few cases in young persons. The remains of old sanguineous apoplexies may be occasionally found on the convolutions, especially near the fissure of Sylvius, whence often the blood has issued.

Tumours of the Pia-arachnoid Membrane.—*Secondary* tumours here as elsewhere have the same character as the primary cancers they arise from. We have met with carcinoma, sarcoma, and lymphoma; probably every kind of malignant tumour would occur here. Thus pigmented sarcoma or melanosarcoma have not infrequently been found widely diffused in the cerebral and spinal pia mater. These tumours invade the cerebral tissue, and are secondary to melanosarcoma of the skin and elsewhere. We have twice known epithelial carcinoma from the face penetrate to the membranes and the brain; other cancers from the bones will do the same.

But chiefly deserving your attention here are the *primary* tumours of the membranes. As fibrous structures these membranes give rise to *fibroma* and *sarcoma*, and this you would expect on the general principle that tumours have a resemblance to their matrix. The sarcomatous growths are harder or softer; they grow inwards into the brain. Giant or *myeloid* cells may be found in them; there may be hæmorrhage into their texture. Virchow has given the name *melanoma* to very rare tumours that arise as an increase of the natural pigment which you find in the pia mater, especially over the medulla oblongata. In two cases this pigment has been found to take the form of numerous small tumours along the whole membrane; in Rokitansky's case this went with similar pigment growth in the skin; these do not invade the cerebral tissue, and are primary. Occasionally you meet with *angeioma* or erectile tumour. One such case is given in the 'Pat

Trans.,' by Mr Morris. They are very rare though. A very interesting form of growth is villous tumour (*papilloma*) of the pia mater; we have twice met with this about the pons. Another interesting tumour is *cholesteatoma*, a specimen of which you see in this bottle. The man from whom it came was in the hospital suffering from cerebral symptoms, and left here to go to Colney Hatch Lunatic Asylum, where he died; this tumour, of about the size of an egg, was found at the base of the brain, loosely connected with the arachnoid, and was sent to us by Mr Tyerman. It was of pearly lustre, and broke to pieces when handled, just as the matter from a sebaceous tumour, with which, indeed, it is in all respects identical, for it consists of a mass of cells containing fatty matter, mixed with plates of cholesterine, and hence the name.

Such tumours are more frequent about the broad ligament of the uterus, and another tumour belonging usually to that region is occasionally met with in the meninges; we mean the *dermoid cyst*. We have met with a hard cartilage-like growth extending from the membranes into the cerebellum. Its structure was like hyaline *enchondroma* in parts, and in parts like osteoid cartilage. It has some resemblance to a curious *waxy* tumour shown by Dr Murchison at the Pathological Society (vol. xiii). We have also met with *epithelioma* of the pia-arachnoid.

Although much less subject to this than the dura-arachnoid the pia-arachnoid sometimes shows small plates of true bone in its substance, as in this specimen over the frontal lobes. You will soon learn that the arachnoid of the cord is curiously subject to osteoid plates. Some instances of small *lipomas* in the meninges have occurred generally about the regions of the brain corresponding to the basi-sphenoid, or on the corpus callosum. One of the size of a bean upon the acoustic nerve is said to have produced deafness.

We have twice met with the small soft cellular tumours under the pons which have been thought to arise from a persistence of the fœtal notochord, the structure of which they resemble, so that Virchow calls them *chordoma*.

VENTRICLES

Inflammation.—*Acute.*—This, in its commonest form, we just now spoke of under "Acute Hydrocephalus." The fact of the ventricles so suffering has given this synonym to tubercular meningitis. Acute inflammation of the ventricles is rarely found except in general meningitis, but we have known cases where pyæmic abscesses have burst into the ventricles and set up a foul and general inflammation within. Dr Delafield, of New York, describes an example of general idiopathic inflammation of the lining of the ventricles. We have in a few cases seen suppuration within the ventricles as a consequence of injury to the head, and in one case a distinct lining membrane of lymph.

Chronic.—Chronic hydrocephalus has a somewhat complex pathology, and probably more than one disease is included under this name. Common as the disease is, yet, being of long duration, the chances are few of our being able to watch a case to its termination, and to examine the parts after death. Those who now and then have had an opportunity of so doing, reporting nothing but increased ventricular effusion, which sufficiently shows that the morbid appearances are not very great; and this has led some to suppose that the disease is in every respect analogous to those examples of pleuritic effusion, where we suppose the membrane to be in a morbid state from the fact of the increased secretion, although there are no changes appreciable to the naked eye. It has also been thought that the effusion might be a dropsy produced by some local inflammatory process, whereby the veins of Galen are impeded. It is probable, however, that further researches may show that the disease owns a variety of causes, for it occurs under such diverse circumstances. Thus, there is the hydrocephalus with which some infants are born; then the more common variety, which comes on some weeks after birth; and there is also another, in grown-up persons, of which we have now seen many examples.

The history of acute hydrocephalus, in which the ventricles are affected by the entering *velum interpositum*, shows that a large accumulation of fluid in the ventricles will arise from active effusion. But the more care we have used in examining the communication between the fourth ventricle and the subarachnoid space the more we have been convinced that an obstruction of this opening is a common cause of effusion into the ventricles, an opinion expressed long ago by Mr Hilton, with whom we had an opportunity of seeing a case of the kind. A gentleman who had never had his mental faculties right since birth, died rather suddenly, and an immense ventricular effusion was found in the brain. In this case the arachnoid appeared everywhere opaque, particularly at the base, and it seemed to close the fourth ventricle. Since this we have had several cases of the same kind. In one the brain was adherent firmly around the foramen magnum, and the membranes and choroid plexus at the infundibular opening of the fourth ventricle were very thick, almost closing the opening. But the most remarkable case we have seen was one in which on opening the skull there appeared a large bladder like a hydatid separating the left cerebral convolutions. This proved to be the immensely dilated lateral ventricle which had thrust its way through to the surface, where it occupied about two superficial inches. The cause of this was found to be a papillose tumour in the floor of the fourth ventricle composed of ependymal structure. It was of the size of a small hazel-nut, and completely cut off the cavity of the ventricle. Two remarkably similar cases are given by Virchow. Again, we have known the ventricles of the brain distended much in a case of pressure on the spinal canal through disease of the cervical spine, thus cerebral symptoms became added to the spinal. Another certain cause of expansion of the ventricles is tumour or tubercle under the *tentorium cerebelli;* whether it grow in the pons or the cerebellum, it compresses the fourth ventricle, and disallows the free escape of the cerebro-spinal fluid. In explaining these results you must remember that this fluid is secreted into the ventricle by the choroid plexuses, and finds its way from them down through the opening of the fourth ventricle into the subarachnoid space.

As we said before, in chronic hydrocephalus the fluid is always within the ventricles; an external hydrocephalus is spoken of, but we never saw it.

A chronic inflammation of the ventricles, shown by a granular condition of their lining membrane or ependyma is sometimes met with. We have already mentioned such a condition in the brain in acute hydrocephalus; but it may be constantly met with in the ventricles of cases where the meninges are thickened. In some such the whole of the interior was covered with large translucent granulations, which once we saw as large as hemp-seeds, giving the surface the appearance of the leaf of an ice-plant. In other cases the change is more local. The substance of these little excrescences is much the same as that of the ependyma, of which they represent warty or papillose states. You will not fail to notice if you look carefully that it is nearly constant to find a few such grains on the *septum lucidum* about the entrance of the vein of this septum into the choroid plexus, these are evidently equivalent to internal Pacchionian bodies.

Blood in the Ventricles.—Blood is found in the ventricles in some cases of apoplexy. The entrance of blood into the ventricles is a grave occurrence in the course of an apoplexy. The blood will sometimes fill the whole of the ventricular system and coagulate there, so that if you remove it carefully under water a mould of the ventricles is found to have been taken most exactly by it. It extends down the subarachnoid space of the cord, though it is not usually followed there, so that this part of its history has been overlooked, still you will always find it so.

In cases of severe injury to the head the ventricular liquid is usually bloody. This you will generally find to arise from rupture of the *septum lucidum.* It is at first surprising that this part of the brain which is protected by its central position and placed in a water-bed should suffer more than others; yet often it suffers disproportionately. Thus we have frequently seen it torn rather freely when little injury was done to the other parts, and once it was ecchymosed after a blow when there was no fracture of the skull. But although the bath of fluid it lies in is very admirably suited to reduce slight vibrations on their way to the septum, yet the water will move *en masse* against the septum when the head is forcibly thrown against an obstacle, if the slightest alteration in the shape of the skull be made in the supreme moment of contact, and thus it becomes very liable to be burst through, as you may often find it, or seriously torn as is more frequent. We have not seen this point noticed, but it forms an interesting example showing how protections from minor risks may themselves operate detrimentally in greater risks.

Closure of Cornua.—The posterior cornu of one or both lateral ventricles is often found closed up, as Esquirol long ago remarked. This is caused by the inward pressure of a deep sulcus of the surface. Sometimes the communication of the cornu with the ventricle is cut off so that the cornu forms a cyst. This occurrence has no pathological significance. Grohe gives an account of three large cysts in connection with a sarcomatous tumour of the brain. He believed these to arise by alteration of the lateral ventricle.

Tumours in the Ventricles.—We have mentioned warty growths from the ependyma in the fourth ventricle. The ventricular surfaces are, however, very little prone to originate either morbid growths or inflammation.

In cases of chronic atrophy of the brain, and in old people, it is not uncommon to find *cystic* formations in the *choroid plexus.* The cysts may be two or three in number, and of the size of peas, or larger, as you see in this specimen. They were formerly called hydatids. Changes may occur in these converting them to *earthy concretions;* these then are found as rounded bodies composed of carbonate and phosphate of lime, and have no analogy to bone. Such stony masses have been called *psammoma* or sandy tumours.

Hydatids occasionally occur; here is a preparation showing two cysticerci which came from the lateral ventricles.

CEREBRUM

Hypertrophy.—We have no special experience of this condition; it is very questionable whether true hypertrophy of the brain exists. We all know that scrofulous children often have large heads; but this unusual size of the head is generally only apparent, being due to the smallness of their limbs, and it does not signify enlarged brain. In cases which have been called hypertrophy this condition was supposed to be the cause of death. Thus, occasionally you hear of a child who has had cerebral symptoms, so that it was expected that hydrocephalic disease would be found; but upon removing the calvaria the hemispheres were seen to be flattened against the skull, or actually protruding through removal of the pressure just as if there was a large ventricular effusion; but on examining the central parts of the brain no fluid was found. On the contrary, the ventricles were small, and, therefore, the size was considered due to an actual growth of the cerebral structure, or a morbid hypertrophy. In cases that have occurred lately the hypertrophy was found to be due to increase of the neurilemma or cement between the fibres. We have ourselves no experience of this state.

Atrophy.—This may be general or local. A *general atrophy* or atrophic induration accompanies and causes the subarachnoid effusion we have recently described to you (p. 204) Thus, in old age, in persons long bedridden by sickness; in drunkenness, lead poisoning, &c., and in old cases of mania, the brain is found shrunken, not filling the skull, the convolutions parted asunder and pale. As to the brain itself its atrophy is seen in the following particulars. The organ is shrunken as already described; its ventricles are commonly dilated, their lining thick and often granular on its surface; the surface of the *thalamus* and, in a less degree, of the *corpus striatum*, is sinuous and shrivelled looking. But it is not only the surface that shows the change, for everywhere throughout the brain you will notice its tissue shrinking away from the blood-vessels, leaving them in wide canals twice or more their own width. This is most conspicuous about the perforated spots, *i. e.* in the lower part of the corpus striatum. The microscope shows in such brains, especially in the thickened ependyma, but also in the neuroglia throughout the organ, the presence of the so-called amyloid or colloid corpuscles, small round bodies of laminated structure like to starch grains. Often, but not always, these are stained purple by iodine, sometimes they are tinted brown, sometimes not at all specially coloured by it. They have always excited great interest through their close resemblance to vegetable starch in their more perfect forms, sometimes they even have the hilus, just like starch grains.

Local wasting arising from diseased blood-vessels, inflammatory softening, &c., is very common. Where the cause is not very evident, we are obliged to be content with the term atrophy. Such cases are occasionally met with where a few convolutions have disappeared, or a yet larger part of the brain may be missing. Mr Partridge recorded a remarkable case of atrophy of half the cerebellum.

In some cases of congenital paralysis, where from birth one side of the body has been atrophied and useless, the opposite side of the brain has been found wasted. This has, apparently, resulted from disease in fœtal life: a theory which receives some confirmation from a case we lately read, of a child who was born hemiplegic, and dying soon after, a clot of blood was found on one side of the brain. This the doctor very naturally attributed to a blow which the mother had received on the lower part of her abdomen during her pregnancy. If, then, the child had grown up, an atrophy of this part of the brain would have ensued, with a permanent paralysis of the other side of the body.

Injury.—Injuries to the brain are of a dangerous character; yet if no general inflammatory action ensue, a considerable loss of substance may occur without destroying life. A case is reported of recovery after the escape of brain tissue through the external ear in fracture of the base. An incised wound may heal, as in any other organ, and, without doubt, slight bruises to the substance are constantly recovered from. We have already given an account of two cases where there was evidence of recovery from severe bruising of the brain (p. 197), such cases would clinically be called concussion. No doubt in most cases of concussion such bruising is the cause of the symptoms. We have not unfrequently had opportunities of seeing brains which have suffered the slighter degrees of concussion; thus, sometimes when death has occurred from other injuries the cineritious structure is found bruised and ecchymosed, although little symptom was observed during life. In more severe cases of injury to the head with or without fracture to the skull, such an ecchymosed condition is always found.

The relation of the bruised part of the brain to the part of the skull that received the blow is of great importance; we will shortly state the law of it. In blows of great velocity and little weight the skull is broken and the brain bruised immediately under the injury in the bone. But in blows of low velocity and heavy weight, as especially in falls, while the skull is fractured at the point struck the brain is always found bruised at a point opposite to this, so that if a man, as usual, falls on the back of his head, rather to one side, the skull is broken at the back and the blood outside the dura mater is always just where the fracture is; but the front of the brain on the other side is the part bruised. The membranes are here usually torn, and you find blood effused in the arachnoid and subarachnoid spaces. The surface of the brain, to a greater or less depth, is bruised up and mixed with blood, while about the bruised part are some conspicuous round extravasations. The bruising affects the grey matter more than the white, even where by the course of the sulci both are removed equally from the surface. It is easy to understand why the brain should be bruised under the spot struck in the sharp light blow; but it is not so easy to see why a heavy blow should smash the brain at a point opposite the seat of its impact, when the skull is always broken at the point struck, while the brain comparatively escapes there. This fact is expressed by the term *contre-coup*. The common explanation supposes that the vibrations in the skull, which radiate from the impact, pass round the sphere of bone, and, converging at the opposite point, are not able to fracture the skull again there (*contre-coup* of the bone is very rare), so they expend their fury on the soft parts, tearing up the brain. But in using this explanation you must remember that the skull is not a sphere, but its bone is rough and has inward projections; again, its base fits at certain parts very close indeed to the brain; you know these parts are the roof of the orbit and the depth of the middle fossa, and if you see a large number of cases of *contre-coup* you will be surprised to find how constantly those parts of the brain which touch the base of the skull at the points named are by far most severely lacerated. The reason of this, no doubt, is that when the form of the skull is slightly changed by its yielding in the supreme moment of impact, the rough base is forced down upon the brain where that is in such very close contact with it. This, you will see, does not oppose the other explanation, but is required in addition to explain the constant relation of *contre-coup* to the anterior and middle fossæ in falls on the back of the head. Mr Hilton, however, pointed out very ingeniously that the ridges of the skull, which appear to adapt themselves to the sulci of the brain, converge and carry the force towards the clinoid process which are near those bruised parts of the brain. We believe this too is an important part in the sum of causation.

Sometimes the deep-seated parts of the brain suffer hæmorrhage in blows on the head. This is very important in medico-legal practice, for after an injury to the head it may be said the person died of apoplexy. To be guided rightly here you must remember that the chief seats of spontaneous hæmorrhage are about the *loci perforati*, i. e. the neighbourhood of the corpus striatum, and that such apoplexies rarely occur without granular disease of the kidneys, or disease of the heart, or aneurism, or other disease of the cerebral arteries; and, again, the effusion is always large, generally two to four ounces of blood. On the other hand, the deep-seated bleedings from injury to the head are usually of small size, we have never seen one larger than a walnut, and that but once. In a case by Casper there were four drachms, but they are generally much smaller, and are generally multiple, so that several of them will be found scattered through the brain if searched for carefully; they have the appearance of little beads of black blood clot, the blood being generally held within the perivascular sheath and thus made to keep a circumscribed roundness. On account of their number these deep-seated extravasations are sometimes distinguished from the superficial as "diffused ecchymosis," the superficial being called "circumscribed ecchymosis." They generally do not occupy the usual seats of apoplexy, though exceptional cases have occurred of effusion into the corpus striatum caused by falls, but then it must be doubtful whether an apoplexy did not precede and cause the fall, as in any such case we should be inclined to believe. By careful attention to the circumstances of each case you will generally be able to come to a sure conclusion, but this is one of the points that try the quality of a morbid anatomist.

No doubt these lacerations of the brain frequently occur in railway accidents, &c. where persons striking their heads violently suffer concussion at the time, and have cerebral symptoms for months afterwards. Judging from the recovery of such persons, and knowing what happens when the head is struck, we think a considerable amount of superficial injury may be repaired. Sometimes, on the cineritious substance of the surface you find spots of a dark yellow or brown colour, and, on minute examination, you discover in these remains of extravasation of blood, with some granule masses, showing that an irritative softening process has been going on. Probably no new cerebral substance is produced, but an indentation or cicatrix finally remains. In greater degrees of injury of the brain we believe that the ventricles are torn open much oftener than is generally taught; Mr Prescott Hewett has collected several cases showing that the lateral ventricle may be opened into continuity with an external wound. The third ventricle is, no doubt, often torn in fractures running through the sphenoid bone; for you know how delicate is the *lamina cinerea*, which closes the third ventricle. Such laceration may lead to flow of ventricular fluid from the pharynx, or if the Eustachian tube and tympanic membrane be fissured, from the external ear, and that even without any fracture of the temporal bone. If the fracture is through the petrous bone and tympanum, the fragments drag upon the pneumogastric nerve; we have observed that this nerve has a close union with the lateral course of the fourth ventricle, so that a pull on the nerve always immediately tears open the ventricle. Thus, if the petrous bone be broken into the tympanum, with a slight laceration of the dura mater, and the nerve tear the ventricle, there is at once a road opened from the ventricle to the external ear, by which the ventricular fluid escapes. You will please not fail to notice that sometimes the jugular fossa is so close to the tympanum that the septum is most easily burst; a common pin will go through it with scarce any effort. This relation has been scarcely sufficiently noticed, perhaps because it is not constant. Sometimes the septum is thicker; you remember the variable size of the jugular fossa. No doubt this is the most frequent source of bleeding from the ear in fractured base.

The severest cases of injury produce *hernia* or *fungus cerebri*, as you see in this specimen, where, after a fracture of the skull and injury or sloughing of the dura mater, an inflammatory exudation takes place, and this, mixed with the softened cerebral substance, protrudes from the surface; if cut off, it is found composed of brain and inflammatory products. Subsequently granulations may form on the surface, and a cicatrix be produced, uniting together the cerebral structure, membranes, and integument ; but too often, as in these specimens you find an abscess under the hernia, causing its protrusion, and ultimately proving fatal. Various other injuries, which may arise from fractured skull, we need not mention, as they are different in every case ; but it may be as well to remember that, as we just now said, you may meet with a laceration of the septum lucidum, and extravasation of blood into the ventricles. You should especially bear in mind the occasional injury to the brain by perforation of the orbital plates, as with a tobacco pipe or umbrella stick ; in such cases there may be no sign of the severity of the injury until meningitis sets in. A fall on the nose may break the ethmoid by the impact of the nasal bone, and so cause meningitis.

Congestion and Anæmia.—These are conditions we have already spoken of in connection with the pia mater; for the brain shares the congestion of its membranes. We will only warn you, therefore, again not to attach too great importance to fulness of the vessels, which implies often merely a dependent position of the head, or that the person has died with some pulmonary obstruction, all the organs alike being full of blood.

Hæmorrhage.—Of hæmorrhage from injury we have just spoken, also of meningeal hæmorrhage (p. 213), and hæmorrhage into the ventricles; we will now consider hæmorrhage into the substance of the brain which constitutes the more common form of apoplexy. First we will allude to capillary bleeding, where the blood is seen bursting forth from various parts of the substance : if a section be made in this state, not only are the *puncta vasculosa* or divided blood-vessels seen, but there are spots of ecchymosis where the blood has actually escaped into the tissue. In the same way, on the interior of the lateral and fourth ventricles these spots may be observed, and amount sometimes here to considerable extravasations into the tissue. This condition of of the brain denotes a diseased state of the blood or the vessels generally, and is found in purpura, idiopathic anæmia, and sometimes in fever. We think it interesting in connection with paralysis, which may come on in cases of fever shortly before death ; we have then found this state of capillary apoplexy to exist. It is often present also after the convulsive attacks occasionally observed in typhus.

But *apoplexy*, as generally understood signifies those cases where blood is effused in some quantity into the *substance* of the brain. We may here remark upon the ambiguity of the term apoplexy; it was originally employed to denote a disease where the patient was struck down senseless, whether this were due to an effusion of blood or other internal cause, and in this sense it is still used by some writers ; but as the cause of such sudden attack was often due to an effusion of blood, the term apoplexy became used by many as synonymous with such effusion, so that it was carried over to express similar effusion of blood in other organs. Without commenting upon the absurdity of this, we must use the terms as they are generally employed, and as regards the brain, to avoid confusion, adopt the term *sanguineous apoplexy* for the hæmorrhage we are now speaking of. It arises from the rupture of a blood-vessel, owing to the strain on its walls becoming greater than their strength will endure. The arteries of the brain are thinner than other arteries, and the substance of the brain is so soft that it but feebly supports them when weakened. Again, we have several times (see p. 207) spoken of the peculiar resistance to the freedom of cerebral circulation which arises through the means taken in the longitudinal sinus to prevent too easy an emptying of the brain by gravitation of its blood. Experience shows that the cerebral vessels will endure the strain upon them so long as their structure continues healthy; but more or less disease of the coats of the vessels is nearly constant after middle life, and is even frequent in early life. This disease show itself in the cerebral arteries of all sizes, but in at least two different ways. The greater vessels about the base and the perforating arteries show chronic deforming arteritis, such as we have already described, but the smaller arteries in the cerebral substance suffer a degeneration which, though essentially similar, has a somewhat different appearance, perhaps only because as the change is microscopic, its detail becomes more conspicuous, and takes a greater share in our description. In these little vessels the nuclei of the outer coat become fatty, and this coat becomes fibrous and dilates: but the proper coats do not widen, so that they run through the dilatations and are separated from the outer coat by some interval. In the years 1866 and part of 1867 we examined the arteries of a portion of the *corpus striatum* in every case, and we found it was quite exceptional to discover complete freedom from this change after the thirty-fifth year of life. Sometimes the whole of the coats are dilated and fibrous, forming indeed small aneurisms, such as were described first by Messrs Charcot and Bouchard. These have lately received much attention as sources of cerebral hæmorrhage, and no doubt they do cause minute hæmorrhage by their rupture; but we think the very general existence of the earlier stages or lesser degrees of this affection in the later years of life has not yet been recognised so fully as it will be. The bleedings from such minute vessels must be small, often the rupture only affects the proper coats, so that the blood passes into the space between these and the adventitious coats, thus forming minute dissecting aneurisms; we believe the "miliary" aneurisms of Charcot and Bouchard are of this nature. Their sections appear as little beads of black clot, perhaps partly decolorised at the surface. Similar little beads are found about great apoplexies, and i softened patches of brain, through a like rupture of the proper coat of small vessels, while the outer coat still holds its continuity; but thes small aneurisms do not of themselves, we think, give rise to grea apoplexies. They are important rather, through the degeneration o tissue that accompanies their formation, and through the accumulatio of the effects of rupture of many of them in softened brain, &c. A disease of the vessels accumulates in the course of life, so necessaril with it the tendency to apoplexy increases with age, and, *cæteris paribus* the older the person the more liable is he to an attack; but the grea predisposing cause is granular disease of the kidney. This is, indeed so frequent an associate of apoplexy that you will be surprised at it almost constant repetition in the cases that occur in the *post-mortem* room. Thus, of the last seventeen cases of fatal apoplexy we inspected fourteen had granular kidneys.

The hypertrophy of the heart in this disease we have already spoke of. We will remind you that this hypertrophy is associated wit disease of the arteries, the nature of which is contested; but the mai point in regard of the hypertrophy, a point which is often overlooked is herein, that the hypertrophy is not generally disabled by associatio with dilatation as in primary heart disease; but it actually augment the pressure in the vessels, a fact which the sphygmograph verifies On the other hand, the hypertrophy in primary heart disease probabl never increases so much as the dilatation diminishes the force in th arteries. Hence it is that the cardiac hypertrophy of granular kidne tends to rupture the arteries, while that of primary heart disease perhaps, never does so; but the arteries are also bad, and we thu have two conditions in granular kidney that would act together t produce apoplexy. The strong heart breaks the weakened arteries It would be expected that so simply mechanical an effect would own simple mechanical cause.

The special seat of apoplexy is where large arteries enter the brai to supply those great masses of ganglionic matter; the corpus striatum and thalamus—which subserve the functions of the extremities. Yo are aware that rather large vessels pass from the carotid and middl cerebral arteries directly into the brain at the so-called *locus perforatu anticus*, a spot just under the ganglionic masses we have named whereas in nearly all other parts of the brain the arteries break into wonderful network in the pia mater from which only very minut arterioles pass into the cerebral substance. A very large proportio of all apoplexies, more than three fourths, occur about the perforate spot in the corpus striatum, or thalamus, or both, or just outside o between them; *i. e.* indifferent to them except so far as they are th goal toward which the arteries in question take their hazardou course. Another rather frequent position relatively to its size is th pons in which we met with five fatal apoplexies in three years, as well as some which had been recovered from. The rest of the brain is in all parts about equally rarely affected, except that apoplexies in the medulla oblongata, and in the corpus callosum, are exceedingly rare, while in the grey matter of the surface they are more common.

Indeed it may be shortly said that apoplexy attends the supply of grey brain matter rather than white, and deep-seated grey matter rather than superficial. An ordinary fatal apoplexy displays a degree of forcible destruction which proves the bleeding to be from a vessel of considerable size; no small vessel would carry the force that is exerted, when, as you often see, two to four ounces of condensed clot are found effused (three or three and a half ounces is a common amount in a fatal apoplexy in the corpus striatum). The brain tissue is then torn in a most terrible way, fragments of it are mingled in the clot mottling its outer part, strands of the fibres are rent across, and their torn ends, half an inch to an inch across, are often plainly seen projecting. In the tissue around the chief effusion many small vessels are torn, and generally in such a way that the bleeding from them is confined by their perivascular sheaths, producing on section those small bead-like knots of blood we have before mentioned. The characters of such a violent laceration across the fibres infer a bleeding from a considerable vessel in these cases, and we do not think miliary aneurisms would produce such an effect. We sometimes see smaller apoplexies, and these usually run in the course of the bundles of nerve fibre, being restrained by the resistance of these. This is generally well seen in apoplexies in the pons which evidently separate the fibres in most instances. The disease of the arteries which leads to the rupture is generally chronic deforming arteritis (p. 149). It is interesting to notice that the parts which chiefly suffer apoplexy are those that chiefly undergo softening. For this, like apoplexy, is not a primary disease of the nervous elements, but an accident to it through imperfections of its blood supply; apoplexy may occur on one side and softening on the other. It is when the grey matter actively serves the limbs and demands a rich supply, that the tribute for all this extra wear and tear has to be paid, the limbs themselves suffering palsy through the stress caused on the vessels by their own excessive demand.

Apoplexies are usually single and are thus distinguished from deep-seated effusions due to injury, but we have thrice seen apoplexies in the pons coexist with great apoplexies in the corpus striatum; on one occasion the apoplexy in the pons was double. In these cases the effusions were evidently simultaneous. Dr Hughlings Jackson has collected several examples of simultaneously multiple apoplexy, in one of which there were four effusions in distinct parts of the brain. These cases are, however, quite exceptional, and all the effusions are in the ordinary seats of apoplexy; we do not think that there would arise serious difficulty in distinguishing between such cases and diffused contusion of the brain, in which usually there is some obvious superficial bruising, and the effusions are not in the usual seats of apoplexy; sometimes, however, the difficulty might throw you back on the history and associated conditions, as in Casper's case, in which, with a considerable effusion of blood in the pons, there was a free arachnoid hæmorrhage, like an apoplexy. In very great apoplexies the effusion is apt to reach the inner surface of the brain at the ventricles, or its outer surface in the arachnoid space.

When the effusion is great death occurs in the course of a few hours or a few days; the shortest time in which we have known an apoplexy to be fatal through compression without shock was forty minutes; in this case the floor of the fourth ventricle was torn up to pulp, and the ventricle itself full of blood; another case, limited to the pons, lasted two hours. In very rare cases death occurs instantaneously, as in a case recorded by Dr Dickinson; we quite agree with him in referring the suddenness of the death in such cases to an abnormally precarious state of the heart through fattiness, &c., of that organ.

When the effusion is less in amount or less critically situated, changes occur of a healing nature. The serum must, no doubt, be very quickly absorbed, if, indeed, it ever appears in quantity; we rather believe that the serum is removed during the progress of the effusion, for we have never seen any serum but always remarkably firm clot. The clot changes as well as the wounded tissue. The clot becomes decolorized, turning pale brown or buff from the surface inwards. Meantime it dwindles gradually away; as it disappears the space it occupied is now seen to be crossed by threads, the remains of vessels &c., that escaped laceration. After many months the clot is all gone and nothing is left of it but some hæmatoidin crystals and grains which remain clinging in the cicatrix, but even these may at last quite disappear. All this while the injured texture is forming a fibrous scar tissue close around the clot, and this may develope well, and produce a cyst-like fibrous sac, the "apoplectic cyst;" when the clot is gone this cyst remains filled with clear fluid, crossed, as we stated, by threads. Here are specimens of such cysts at one, two, three, four, and eight years after the apoplexy. The wall of the cyst is made of wavy connective fibre including blood-vessels; it is believed to arise from the neuroglia by proliferation.

Sometimes matters take a less favorable course, and the tissue around the clot undergoes subinflammatory softening, perhaps with fresh effusions of blood; the texture then, for a distance around, is found permeated by those "granule globules," of which we shall speak under Softening. It is said by some, as Niemeyer, that abscess may form at the seat of an apoplexy; we know no facts to support this statement. It certainly is remarkable that severe spontaneous injury to the brain should so very rarely indeed excite the higher inflammations, when traumatic hæmorrhage so often causes suppuration.

The end of the most favorable cases is, then, that the clot is all absorbed, and the cyst-like sac which surrounded it is found with a little fluid in it, or almost or quite closed, but not with its walls adherent. There is danger of recurrence of the accident, and it is far from uncommon to find three or four apoplectic patches in various stages.

We must now notice that some of the worst apoplexies are due to relatively large aneurisms of the greater cerebral arteries, such aneurisms reaching the size of a pea or horse-bean, or even becoming as large as a cherry. We have already spoken of these aneurisms (see p. 159). It is interesting to notice that they are apt to occur in young persons; about half have occurred before the age of twenty. It is highly probable that in young persons, where there is no general disease of the vessels, if the apoplectic portion of brain were thoroughly examined, an aneurismal condition might often be found, for such has sometimes been discovered when not expected. They are far from very infrequent. Thus, Sir W. Gull collected sixty-two of them, and numbers have been recorded since. Here are three which we have mounted in the last two or .three years. They generally affect the middle cerebral artery, or others of the great arteries on the surface of the brain; but Dr Bristowe has given a remarkable case of a large aneurism of this kind within the cerebellum. We have seen carotid aneurism in the cavernous sinus burst into the lateral ventricle.

Besides these greater apoplexies, you will often meet in old people with bad vessels, and especially if they have Bright's disease, with examples of numerous, small, brownish, soft spots, showing the remains of old effusions, &c. Such patches are the causes of slight numbnesses and weaknesses of the limbs in these people.

The Brain in Epilepsy.—It may be convenient that we should now place as a contrast the condition of the brain in epilepsy. We should say there is no proper morbid anatomy of epilepsy. You may find the cause of epileptiform seizures to be some condition irritating the brain, especially changes on its surface; the irritation of such changes having aroused the fits. But this discovery is to finding the cause of epilepsy, like the discovery of membranes on the larynx to finding the cause of coughing. The immediate cause of epilepsy leaves no morbid anatomy. By a series of careful researches, Kussmaul and Tenner were led to conclude that anæmia of the brain is the cause of convulsion. The thoroughness of their system of experiments entitles their opinion to respect. Some, following them, make a theory that the arteries of the brain go through spasms, causing the spasms in the limbs—spasm for spasm; we cannot say as much for such a view. Kussmaul's opinion, however, will not account for the truly rare, yet certain examples of epileptiform convulsion in the height of a paroxysm of emphysema cough, when the face is black with congestion. We may fall back on the subtle ingenuity of Mr Simon, and say that stasis is the equivalent of anæmia; and, perhaps, if we practically do so, and say that the cause of epilepsy is a negative state of blood influence, due either to deficiency of its supply or to inefficiency of its quality, we shall have a simple view, and useful so far as it leads us to cease regarding epilepsy as a strong and positive state.

The so-called *serous apoplexy*, or water-stroke, is a disease of which we know nothing; it was a term formerly used to designate those cases of very speedy death with coma where no blood was discovered in the brain. It was the occurrence of these cases that induced Abercrombie to suppose that the fatal pressure in apoplexy is always within the vessels, and that the escape of blood into the brain is only an accident of some cases. This view he held to the end of requiring all cases alike to be equally bled. But there can be little doubt that the majority of such cases were instances of Bright's disease, and that death was due to what is now called uræmic intoxication; if you read the cases given by Abercrombie of the state of health of his patients, and all the accompanying symptoms, it can leave no doubt on your mind that many of them were instances of Bright's disease. Apart from such, however, we do occasionally meet with cases of very rapid death from evident cerebral affection, and yet scarcely anything is found to account for it on examination; but in these there is no increased effusion, so that the term serous apoplexy cannot be used; it is a term we never adopt, nor do we know to what class of cases it is applicable. Some describe, with Niemeyer, an acute œdema of the brain in Bright's disease, that is, an effusion of serum into its mass from unknown causes, but we have not found anatomical evidence of this. The death in these cases is probably chemical, and not mechanical. It is possible that some instances may have been of a kind such as we have mentioned under chronic hydrocephalus; where, for a long time, a chronic inflammatory action has been going on in the ventricles, and yet death occurred suddenly, and very little was found except the increased fluid in these cavities, which had no doubt existed for a long time.

Inflammation, Encephalitis or Cerebritis.—This may be diffused or local.

Diffused inflammation.—This probably happens much more frequently than we recognise after death. We have already, when speaking of meningitis, both traumatic and idiopathic, said that the surfaces of the brain and the ventricles are undoubtedly affected, and it is highly probable that very often the whole substance is also. Sometimes after injuries we have found large tracts of the cerebral substance yellowish and watery, and reddened in parts with effused blood, at the same time being softened, so that a stream of water washed it too easily away. These cases showed encephalitis as certainly as could be, short of formation of pus. We shall have occasion to mention to you again the difficulty there is in recognising in the solid organs those inflammations which correspond to non-purulent catarrhs of epithelial surfaces, not to speak of urticaria, &c. In the lung (which we are so very falsely apt to assume as a type of a parenchymatous organ in its inflammations, when, in fact, it is not parenchymatous at all), in it there are spaces to receive the readily forming epithelial and effused products of a feeble inflammation; but in the brain there is no space for the products, and no epithelium to form them; yet there is not any reasonable doubt that the brain tissue is liable to inflammatory irritations of lower intensity than the suppurative, parallel to that creating a catarrh, but without its products. However, there is no *post-mortem* evidence of excited states that are not productive. If there were such a change as urticaria of the brain you would not see it after death, any more than you see urticaria, &c., of the skin after death; sometimes you see cases that strongly suggest urticaria of the brain, though that may sound a little like nonsense at first. Thus, not long ago a middle-aged man came into 23 Philip Ward in a state exactly resembling grave apoplexy; instead, however, of dying he got quit from his brain symptoms by the next day, and as he awoke to consciousness his skin burst into the most extravagant urticaria you ever saw.

Perhaps the principal reason why we do not recognise cerebritis is that it is fatal before those changes have occurred which would be apparent to observation. In what is called acute mania, where the illness is of a few days only, very little change is observable, although there can be little doubt that a change, be it only a molecular one, has taken place. We do, however, occasionally meet with cases where, after a few days' illness of a cerebral kind, the brain presents points of red softening and inflammatory product, indicating that a general cerebritis has occurred. In one or two such cases small discoloured spots were found throughout the whole brain, both in the cineritious and medullary substance, and these were found to contain softened tissue and inflammatory cells, sufficient to show that the disease from which the patient suffered was one involving the brain very generally. Nevertheless, we must say that the substance of the brain is exceedingly little prone to spontaneous general inflammation of a kind decidedly recognisable after death. In *cerebral rheumatism*, of which we have examined many examples, we never found any trustworthy signs of an inflammation, though some authors have described an effusion of lymph in that disease. We do not at all mean to say that this proves there is no inflammation, but that, if at all, it is of that low degree which, like many cutaneous eruptions, leaves no trace. However, the hyper-pyrexia, which almost invariably accompanies it, is sufficient to account for the cerebral symptoms without the assumption of inflammatory action. There are few consciousnesses that do not prove volatile at 105°, and in cerebral rheumatism the temperature reaches 110°, or even higher; temperatures at which, according to some authors, a scum would form in the blood. Again, in the delirium and convulsion of fever, some authors have discovered embolic masses in the small cerebral vessels, with a view to accounting for the disturbance of the brain; but we have seen such appearances as those depicted in numerous cases where there were no cerebral symptoms. In rheumatism and the fevers, we believe, the cerebral disturbance is due to high temperature and altered chemistry, and is not anatomical; at the same time our experience coincides to a certain extent with the view which holds that misshapement of head, old alcoholic thickening of the membranes, &c., will make a part of the whole sum, which results in the delirium or convulsions; but in many cases no such anatomical predispositions are discoverable. Often the predisposition is plainly physiological in the sanguine excitable temperament of the individual.

Chronic diffused cerebritis.—Sclérose en plaques.—Insular sclerosis.—This is found throughout the brain and spinal cord, or more rarely limited to either. It was figured by Cruveilhier and Carswell though its nature was not recognised by them. It forms a disease that year by year proves more evidently to be capable of certain recognition clinically. It appears in the form of circumscribed patches, generally circular on section when small, but growing more irregular as they enlarge to the size of a hazel-nut or larger. They have very much the appearance of grey cerebral matter but are tough and firm, the whole brain also being usually much firmer than natural. When small and circular they often appear to surround a congested vessel, but as they grow larger this is not evident, the vessels in them are, however, always rather large and full of blood. They appear to us to arise at small points and spread excentrically like an eruption; when small they have a dark grey colour, as they enlarge they grow paler, until at last they are with difficulty distinguished from the surrounding structure, but they have a slight ochrey opacity and slightly curdy appearance, different from the creamy pinkish tint of healthy brain. They very rarely invade the grey matter, when they do the cells of grey matter persist longer than the white fibres. By the microscope you find within their confines that the nervous elements are greatly wasted away, and a quantity of subfibrillar hyaline material is present, in which are countless granule masses (see p. 235), and often also a variable number of amyloid corpuscles (p. 218). Sometimes this disease appears in one or more larger patches, but this is rare. The extent of its distribution in the crura, the pons, and medulla oblongata is sometimes such as to render it very astonishing that the sufferer could have survived so long; we have seen the pons almost entirely sclerosed to a grey mass. The change sometimes surrounds the aqueduct of Fallopius in a very curious manner.

For a comparison of this change with grey degeneration, see p. 250.

Local cerebritis.—Acute inflammation is seen at a more advanced stage when local, for the remainder of the brain being healthy, the disease can advance to a considerable extent before it puts an end to life. We have already said that such inflammation may arise from apoplexy, but this is generally rather a mere softening or atrophy; on the other hand some of the most marked cases arise through injury to the head. A portion of brain may then be seen of a red colour, intermixed with yellow; the former colour is due to vascular congestion and perhaps extravasation, and the latter to an inflammatory serous effusion mingled with the brain structure. On microscopic examination in this stage you will often be surprised to see how little change there is. The elements of the tissue are softened and granular, but still retain their form, and nothing in the way of characteristic inflammatory elements can be perceived. Such a degree of inflammation is probably recoverable. It constitutes *red softening*, it is the inflammatory form of that condition (p. 234), and the form which is the better characterised of the two states that come under red softening. Our experience is that this inflammatory red softening, if not due to injury, is due to inflammatory embolism or to incipient pyæmic inflammation, as we shall presently mention; when speaking of embolism we have already fully described the means whereby emboli from seats of inflammation start inflammation in the parts they reach (p. 172). The brain furnishes some of the most striking examples of these inflammatory changes. When an embolus comes into a cerebral artery from the ulcerated endocardium you may find the part of the brain that should be fed by that artery in any stage of inflammatory change, such as red, soft, and swollen, or stiffened by a process of semi-induration through intermixture with nucleated fibres of organising lymph, or softened into ochreous pulp, or even formed into an abscess. The latter incident is rare, but we have now met with several instances of true suppuration consequent on this accident. Sometimes the patient survives so long that the inflamed part of the brain undergoes regressive changes. The red softened tissue turns brown, and then ochreous yellow, from change in the pigment of effused blood, for more or less ecchymotic effusion of blood is constant in inflammatory red softening. The tissue may be found broken down; a cyst-like thickening of the surrounding tissue may enclose it, and the contents of this may be in pulp, or even at last a nearly clean fluid crossed by threads of persistent tissue; or it may become the seat of apoplexy.

We have already mentioned that, although it is advantageous to study the diseases of the parts separately, yet we find constantly that all the structures within and including the cranium may be inflamed together; thus we had, not long ago, in the hospital two such cases, which resembled each other in every respect, where the patients were ill with obscure cerebral symptoms for several months. In these the calvaria, as you see here, had been inflamed, the whole internal surface being covered with minute bony granulations, the membranes were also affected, and the brain throughout its structure showed points of softening, which in one or two places became large cysts, as you see in this drawing. These were full of fluid, lined by tolerably good membrane, with membranous partitions and fibres running across their cavity.

Abscess.—Another result of cerebral inflammation is suppuration; this, however, cannot be looked upon as the ordinary extreme of inflammations from common causes. These, as we have just shown, result rather in indurations from exudations of lymph, or softenings, or cyst formation. Abscesses of the brain arise either through suppuration of the bone due to injury or disease, or else they are part of a general pyæmia; as to this you must observe that they may be secondary to suppurations in other parts where the general pyæmia is otherwise slight and transient. The cerebral abscess may be the only secondary suppuration present after an operation. Our own opinion agrees with that long ago advanced by Sir W. Gull, who concluded that abscess of the brain does not occur spontaneously. We have twice seen examples of the occurrence of abscess of the brain from injury without fracture, as mentioned by Mr Prescott Hewett, in these cases if the abscesses should become encysted, as they sometimes do—look at this one which came from a case of fracture of the skull eleven weeks after the injury, when the fracture had healed—the blow on the head might easily be unreported and so the abscess appear to be spontaneous. In any case of abscess of the brain, suspected during life or found after death, you should examine scrutinously for some primary seat of suppuration. We shall impress you with this best by an example or two. In our last cerebral abscess case we were nearly in despair of explaining its origin, having searched in vain all ordinary situations of suppuration, but we found that the right bronchus, from near the end of the trachea to the second bifurcation, had its mucous membrane ulcerated away, so that the cartilages were in parts exposed and necrosed. Now the cerebral symptoms had masked the pulmonary in this case, and it would have been easy to have passed by what was certainly the prime cause of the abscess. We have known abscess of the brain arise from ligature of a pile and from excision of the eye-ball. We have never ourselves met with an example of non-traumatic cerebral abscess where a careful search has not at last revealed the presence of primary suppuration in some other part of the body, and we should consider such a case to be an unsuccessful examination. Next notice that the first stage of the abscess appears as a deep-coloured red patch of softening, such as you see in this drawing. This stage is not often met with, the evidence of the nature of the red patch was its association with general pyæmia. After the development of pus the abscesses will differ according as you find them recent or old; if recent the abscess wall is irregular, or even ragged looking, and has no proper membrane, its contents are more liquid and of a pale-greenish sulphur colour, it may be fetid and black from sulphuretted hydrogen; this depends chiefly on whether the bone disease it arose from is necrotic; an older abscess has a round shape and smooth wall, which is defined by a "cyst" of adventitious connective substance. This cyst may show microscopically good wavy fibre in its middle or best developed part while its outer part shades off into the brain-tissue around, and its inner surface has the character of pyogenic membrane. A very old abscess, which had existed eighteen months, had its cyst so thick and firm that we could lift it out of the brain-tissue around. The man had had pyæmia from lithotomy in this hospital eighteen months before; he recovered and had worked for sixteen months in the fields, when he came in again and died of coma after a convulsion. The contents of these old abscesses are green, alkaline, and even ropy; the microscope shows the pus-cells to have degenerated almost entirely away in old specimens; they may last very long. In one case we knew, there was every reason to believe that such an encysted abscess had been present in the brain for many years. This prolonged innocence and the gradual degeneration of the abscesses make it probable that some of the old calcareous relics found occasionally in the brain may have been due to former abscess. These old abscesses were single in each case, but sometimes, especially in acute pyæmic cases, there are several or even numerous abscesses. Multiple abscesses are generally speedily fatal, but we had one remarkable case with Dr Forshall, of Highgate, in which there were more than twenty encysted abscesses in the brain of a lad who had worked as a shorthand writer to within five days of his death. Abscesses are by far more often found in the white matter than the grey matter of the brain. You remember that apoplexy chiefly affected the grey matter, especially when deep seated. Drs Gull and Sutton say that of seventy-four cases collected by them only four occupied the corpus striatum or thalamus. Abscesses belong mostly, indeed, to those great masses of white matter which constitute the *centra ovalia*, whose function is shown in softening and tumour to be less indispensable, hence abscess is so frequently latent; a man with ounces of pus in his brain may continue to do good mental work and have full use of his limbs.

The greater number of abscesses are produced by old caries of the ear, what it is that superinduces the abscess on the years'-old caries is often not at all clear, but there may be obvious recent extension of the bone disease producing necrosis of the petrous or mastoid bone, so involving the dura mater and sinuses, sometimes the dura mater is not involved. The abscess usually lies close to the seat of its cause, whether this be injury or caries, but a layer of brain-tissue often, not always, intervenes. This neighbourhood is important to remember, it has even allowed the successful evacuation of pus by operation in cases of fracture, and sometimes the abscess near the ear has discharged through the temporal bone (otorrhœa cerebralis). We have mentioned the occurrence of abscess under and causing *hernia cerebri.*

Lastly, the tissue around an abscess of the brain for a greater or less extent is generally softened, from a state of sub-inflammation.

Softening, or Ramollissement—Mollities Cerebri.—This term is in constant clinical use in a very vague sense, as denoting a condition of mere imbecility, or weakness of mind. Such a clinical use of the term refers practically to that white softening of the brain which is the true and primary softening *par excellence.* The other conditions in which the brain is softened are different and fall under other heads of consideration; thus, *red* softening is either incipient inflammation, which has been already sufficiently considered, or else it is embolic congestion, which we will speak of presently. *Yellow* softening is either this very same true white softening, tinted ochreous with decaying blood, or else, and more properly, it is a very different and always secondary condition in the form of a yellowish gelatinous œdema around tubercles, tumours, or syphilitic disease, &c., of the brain. Rokitansky, who describes this latter yellow condition very carefully, says the yellow softening is always accompanied by acid reaction, but we have found it alkaline several times. He says he has met it as an independent change, our own experience agrees with that of Cruveilhier that it is always limited to the immediate neighbourhood of growths to which it stands in the relation of a peripheral œdema. There is no serious microscopic change in it, only excess of fluid; it is of little importance. Even *white* softening is ambiguous; for besides the true white softening we are going to describe there is that totally different sodden white softness of the central cerebral tissue around the ventricles in acute hydrocephalus, which we have already described (see p. 211). This is little more than a mere œdema and is devoid of the special characters of true white softening.

Reviewing then these softenings, which are to true white softening as birds that are black are to blackbirds, and noticing them as (1) inflammatory and (2) embolic *red* softening; (3) the *yellow* softening around tumours, as well as (4) the œdema of the central white matter in acute hydrocephalus, there remains that white softening, occasionally tinted yellow, that is meant when we clinically diagnose "softening of the brain," or *ramollissement*, or *mollities cerebri*. We will now confine our attention to the characters of this, as it has a speciality and independence which the other softened states do not possess, for they are all, as you see, dependent parts of otherwise recognised changes. This true white softening can be generally easily recognised by the loss of cohesion, and pastiness of the brain matter; but if not, the fall of a gentle stream of water will detect the change. There is, we think, no case where the microscope can be put to such good use in morbid anatomy as in identifying this softened cerebral structure, for we not only find the tissues broken up, but mixed with them a number of bodies known as granule masses, and which you cannot mistake. These are large bodies, appearing black by the microscope, from not transmitting light, and sometimes compared to mulberries, from being composed of a number of round granules; their principal composition is fat. It was once thought that such bodies denoted inflammation, and they were called Gluge's inflammatory globules, after the projector of this view; but you may find bodies essentially similar in any degenerating part, as a decaying strumous gland, or a cancerous tumour, or a phthisical lung; how they are formed in the brain is not yet quite clear, whether they originate in inflammatory cells, or are the natural cells of the cerebral structure degenerated. In some you may still see a wall and a nucleus so bold and manifest as to point to the former opinion as the more correct; be this, however, as it may, you can at once recognise these large dark bodies in the cerebral structure, and, once having done so, you know the part is diseased or softened; you want to ascertain, in fact, whether a part of the cerebral structure is healthy or diseased, and the discovery of these bodies at once decides for you; and it is for this reason we should advise you to make yourselves familiar with the appearance of softened brain and medullary structure. Besides these bodies the microscope in soft brain will show decaying tubes and escaped myelin, a few amyloid corpuscles from the neuroglia, and the fatty and calcareous remains of blood-vessels.

Now as to the cause of this white softening it was always supposed to be due to a chronic failure of blood-supply on account of disease of the vessels, but there has been a good deal of question of this lately since it has been shown that obstruction of the vessels produces a *red* and softened state of the brain, and various views are held as to the conditions in the vessels which induce the red or white condition of softening respectively. Some think the white succeeds the red as a stage after the redness passes away, but as red softening always includes some effusion of blood, no doubt its subsequent stage is represented by those ochreous softenings which we were just now alluding to as tinted with decaying blood. This ochreous state is, however, so near to our white softening that distinction begins to vanish. Others think that to produce white softening there must be, besides the obstruction of the vessel of the obstructed part, some conditions of the vascular supply around it preventing reflux into the stagnant region, but it is not easy to see what condition of vessels this would be. Dr Delafield lays it down that the difference is one of extent, embolism of small arteries leading to congestion from parts around, while obstructed great vessels do not become so easily refilled; but in cases of ligature of the carotid we have found the corresponding hemisphere swollen and red, so this cannot be the explanation. We believe the difference is due to the difference in degree and rate of the obstruction of the vessels. The softening is white when the obstruction is slow, so as to starve the tissue without inducing absolute stasis of the blood and subsequent hyperæmia; thus, slow syphilitic inflammatory stoppage of the arteries has led to extensive white softening of the pons and posterior lobes of the cerebrum.

Again, it must be remembered that the embolic red and the white softening occur in very different classes of cases. In cases of ligature of the carotid and in experimental embolism, as in Prevost's injection of solid substances into the carotid of dogs, from which the history of embolic softening is generally drawn, the circulation is vigorous and the blood plentiful; but white softening occurs in old persons who are poor blooded and have very enfeebled circulation. When we were speaking of embolism and its effects (p. 171) we showed you that the congestion which ensues in an embolised spot is due to the general tension of the vascular system forcing blood into the part whose vessels have lost their tonic resisting power through mal-nutrition, so that the lowering of the tension in enfeebled age would prevent its occurrence. In short, *red* embolic softening occurs when the vascular tension is high and the obstruction sudden and complete, while *white* softening occurs when the vascular tension is low and the obstruction gradual and less complete. We will refer you now to the general remarks on the effects of embolism for an explanation of the red softening that ensues on the occurrence of embolism in the brain. We have seen softening of both sides in the same brain from embolism of both Sylvian arteries.

Syphilitic Disease of the Brain.—We have given some cases of general meningitis which occurred in syphilitic subjects. These are rare and indeed some doubt will perhaps attach to them until the observations are more extended. We will now speak of characteristic syphilitic gummatous disease of the brain. Quite contrary to the meningitic cases we were alluding to, the peculiarity of this form of disease is its circumscribed extent. It always appears as a tumour-like nodule which is generally single, though occasionally there are several. It by far most commonly affects the surface of the brain, so that it is quite rare to find syphilitic disease of deep parts unconnected with the surface. Virchow makes the interesting remark that the parts affected are those most prone to suffer in mechanical injuries to the head as though the effects of such injuries predispose the part to suffer. The syphilitic gumma has generally an irregular figure, representing indeed usually the form of the part attacked and changed by it. The consistence is firm, the colour pinkish red, like pale flesh at the outside, with a caseous centre which represents the older decaying part of the growth; or this caseous part may be in the form of a mottling of the central area through coalescence of its originally distinct patches. But always the caseous portion forms the greater part of the patch; the tissue around is commonly in a state of yellow softening, but the patch has no defined surface towards this.

It usually, by being formed in the pia mater, causes adhesion between the membranes, and thus the dura mater is found fixed to the brain. On the inside it involves the grey substance. A very favorite seat is about the Sylvian fissure, and here it invades or obstructs the middle cerebral artery. This accounts for the paralysis which accompanies the convulsive fits set up by cerebral syphilis.

It is far from easy to distinguish these patches from tubercle on the one hand, and from sarcomatous tumour on the other. But a cerebral tubercle is generally deep seated and has a spherical form; it is more distinct from the parts around than the syphilitic growth. It is more entirely caseous, and its cheesy part has a greenish hue and a more curdy aspect; the margin of vascular tissue is very narrow. In each of these points you will see that there is a distinct contrast with the syphilitic gumma.

From tumours syphilitic gummata are known by the larger proportion of caseous change they have undergone, as well as by their less defined surface, and by the less perfect development of their elements. In syphilitic formations the cells are always ill-bred looking, short and small; spindles and round cells with granular unclearness and imperfect nuclei; while in sarcoma and myxoma of the brain, it is remarkable how beautifully developed are the individual cells in the undecayed part of the tumour.

Induration.—Except the insular sclerosis of which we spoke (p. 230) and the firm state of the brain in old wasting (p. 218), we do not know of induration of the organ; we have found it firm in typhoid and typhus fever, as Rokitansky remarks, but not "indurated." Indeed a general induration, involving the whole brain, we have never yet met with, although such a state is described. Dr Bright, for instance, in his *Medical Reports*, case xix, relates the case of a little girl, who, during a whole twelvemonth, had been imbecile, and had contraction of the limbs. After death, the brain was found shrunken, and the medullary part almost as hard as cartilage, so that when the cortical portion was removed it remained like a complete cast of the convolutions. The case, however, corresponds closely with diffused sclerosis, and may have been of that nature.

Morbid Growths.—The list of cerebral tumours is a list of tumours generally; for every named kind of tumour has been found in the brain. Our notice of each must then be short and limited to those points special to the brain. (For the histology of the several tumours see the Plates 1 to 6.)

Carcinoma.—This we have found in a most typical form in the brain, secondary to scirrhus of the breast, &c., its cells are apt to form glomerules in a more hyaline and less fibrous meshwork than that found in the breast and other parts. This reminds us that *myeloid* tumours have been described as present in the brain, several such cases are in the Pathological Society's Transactions. This drawing shows the structure by which the myeloid nature is suggested. There are large "giant cells" in a semifluid or even fluid pulp, such tumours resemble abscesses, but the apparent pus is not green, like true pus in the brain. When these giant cells were supposed to characterise a particular form of tumour called "myeloid," it would be natural to call these tumours myeloid, but no form of cell is now held to characterise tumours, and giant cells are found in almost every kind of tumour, even in tubercles. The tumours in question are very soft carcinomata.

Sarcoma.—You meet with very beautiful spindle-cell tumours in the brain, the spindles peculiarly long and well formed, and easily extricated from their neighbours, such tumours are generally encapsulated, the capsule closely resembles pia mater, and often is connected with this membrane. Hæmorrhage into these tumours is frequent.

Myxoma.—These occur not infrequently. According to Virchow heteroplastic myxomata form very often in the nervous tissue; he associates this with the close alliance in nature between the soft neuroglia and his mucous tissue. They form large tumours, of a soft translucent gelatinous substance, and by the resolution of their central part they may break down into cyst-like cavities full of slimy fluid. He gives instances in which they appeared to be congenital.

Melanosis of the brain has occurred occasionally as part of a general melanotic disease; it is very striking because of its black colour against the white of the brain. But, as we have said before, melanotic cancer is only ordinary cancer pigmented, but otherwise just as usual.

Glioma.—This is the proper tumour of brain tissue. It is distinguished from the others by having no capsule, but merging indefinitely into the tissue around. It is firm and tough, otherwise very like brain tissue, but more pellucid. It is very liable to undergo successive hæmorrhages, so that blood in various states is present in the tumour, giving brownish or purplish colours to the central parts of it, or ending in false cysts there. The microscopic structure is very variable. Sometimes there is scarcely a trace of structure except fine, delicate filaments forming a feltwork, then you get cells small or large, scattered in such a tissue, resembling sarcoma very closely (and being indeed the sarcoma of brain). We have twice met with a very peculiar form of it, in which the cells were like those star fishes that are called "heads of Medusa," each having a beautiful conspicuous cellular body, with countless bushy stellar rays running out of it, very like those of a bone-corpuscle. The tumours in these cases were scarcely discoverable by their colour, only by their consistence and the pinkish clear swelling of the affected part. It is to these tumorous swellings or "infiltrations" of parts that the question must always apply whether the tumour is not due to a chronic inflammatory irritation, whether, in short, it is not a chronic inflammatory thickening of the brain substance. Often you find such a question not easy to solve.

Osteoma.—This has occasionally occurred in the brain. Such bony tumours are of slow growth and may be almost latent. Thus, Ebstein fully described an osteoma of the cerebellum which had produced no symptoms.

Heterotopia cerebri.—We have only once met with a decided example of this curious condition, a development of true ganglionic grey matter under the ependyma of the lateral ventricle (posterior cornu), of the size of a hazel nut. Attention was directed to these first by Rokitansky and Virchow; they describe multiple patches in the same situation.

Erectile tumours.—We have ourselves never met with these. Cases are recorded; we have mentioned Mr Morris's case of angeioma of the pia mater invading the brain.

Cystic tumours.—These are very rare. The recorded cases are few; we have here a remarkable cystic tumour of the cerebellum. The cyst is as large as a small apple; the little mass on one side of it is well-formed carcinoma, and the tumour was primary. It is curious that cysts should occur at all in a part like the brain, which possesses no elements whose dilatation could form them. In this case we quite clearly traced the cysts to the widening of spaces caused in the tumour by extravasated blood. We have before alluded to the posterior cornu of the lateral ventricle cut off and forming a cyst. Dermoid cysts have occurred in the brain.

Calcareous masses are met with sometimes in the brain, and are probably old decayed tubercles or dried up abscesses.

In thinking generally as to any question of the malignancy of a cerebral tumour, you must remember that we know the malignancy of a tumour most certainly by its infecting other parts, as the lymph glands or viscera. Now the brain is so vital an organ that death may be thought likely to supervene before time for secondary growths has elapsed. Again, doubt and obscurity still hang about the distribution of the lymphatics within the skull, it is even questionable whether there are any lymphatics there at all. We must say, in spite of the description of lymphatics within the skull, that we have never found the lymphatic glands at the base of the skull enlarged in cases of malignant tumour of the brain, unless such tumour implicated the dura mater or bone. Thus it appears that there is not the usual channel for invasion of the glands, and hence the ordinary generalization of cancer is thought not to occur in the brain.

Tubercle.—We have already spoken of tubercle affecting the membranes, and of its being especially found in those portions of the pia mater which dip down between the convolutions. When tubercle occupies the brain tissue it rarely appears in this form, but in large yellow masses, resembling milk of sulphur in colour; these are always very firm, and little disposed to break down. Occasionally we have seen one softened in the centre, but considerable softening of them is rare. These masses, from growing in a soft organ, are generally quite round; and thus a globular tumour the size of a billiard ball, or even larger, may sometimes be found. Then their surface you see by careful scrutiny to be made up of agglomeration of minute miliary tubercles which spread into the tissue around, while the advancing caseous mass involves them in its spread. Rindfleisch speaks of two forms of these tubercles, one of which is thus made up of miliary aggregations, while the other is only an almost amorphous scrofulous deposit; we believe this difference is one of age and activity, and that the latter form is an older tubercle, not spreading at the time of death. We mentioned that these tubercles sometimes set up tubercular meningitis. We believe the larger tumour of "tubercle" in the brain is the primary tuberculosis or "scrofula of that organ, while the miliary form is always secondary, or, at any rate, is part of an acute tuberculosis. Miliary tubercles in the substance of the brain must be very rare, for we have only once met with a few of them. Dr Gee, however, describes a case where part of the cerebrum was softened and stuffed with minute tubercles in tuberculous meningitis.

These large scrofulous or tuberculous tumours are more frequent at the base of the brain than elsewhere, and thus are very commonly met with in the cerebellum, which is their favorite seat. Sometimes, on removing the brain, a lobe of the cerebellum may appear, slightly altered in shape or size, and then, on cutting it through, a large uniformly smooth yellowish mass is seen occupying this part, but having a layer of cineritious structure over it. Tumours in this neighbourhood press upon the surrounding parts, and produce various symptoms; amongst others, cause ventricular effusion, and give rise to conditions resembling those of chronic hydrocephalus. We can call to mind two cases of chronic hydrocephalus where these large scrofulous masses existed at the base of the brain.

It is truly astonishing how great a destruction of the vital parts of the brain may be insidiously affected by these chronic large tubercles. Here is a *pons Varolii* with a tubercle occupying its central part to such an extent that the remaining tissue forms only a delicate, scarcely continuous film around it. Sometimes they reach an enormous size. The preparation, like a large potato, which you here see, was presented by Dr Hughlings Jackson; it is from a cerebral hemisphere. Sometimes there are more than one tubercle present, but generally one only.

The microscopic structure of these tubercles is seen at their peripheral part; you here find often a concentric arrangement of lymphoid cells around vessels, the whole representing a considerable degree of structure. But within a few lines of the surface inwards the elements are faded away to relics. Outside is a red zone of soft tissue, often very delicate, so that the tubercle easily falls out. For an inch or so all around, the brain tissue is in a state of yellow softening (Rokitansky's).

Hydatids.—We have mentioned these as occurring on the surface of the brain in connection with the arachnoid, and also in the ventricles. These have always been, we believe, *cysticerci*; but the *echinococcus* is occasionally met with in the substance of the brain, as you see in these old specimens. We recently had a specimen of a very large echinococcus in a young girl from the clinical ward. This reminds us that it is said the echinococcus occurs more often in early life, and the cysticercus in older people. When one is present in the brain, another echinococcus is usually present in the liver; it was so in our recent specimen, but it is not always so. Generally they are acephalous; but scolex heads are sometimes present.

PITUITARY GLAND, OR BODY

This may undergo morbid changes, and be subject to diseases, as other parts, and thus more than once have we seen it occupied by cancer. Owing to the large cells which the glandular part of this body contains, with their numerous nuclei and pigment, it was suggested by

Dr Gull whether this body had any relation with the supra-renal capsules, which contain somewhat similar cells; we have since looked in all cases of morbus Addisonii for any disease of this body, but have not yet found any appreciable change.

The PINEAL GLAND often contains calcareous sand. This, called brain sand, *acervulus cerebri*, is generally seen in grown persons along the anterior border of the gland and its peduncle (*psammoma*). We have several times found cysts in this gland, once as large as a cherry, and once with a hæmorrhage into the interior. No symptoms were evidently referable to these changes in the "gland." Two of these cysts occurred in Bright's disease.

SPINAL CORD

We will consider the diseases of the membranes and medulla in succession, in the same way as we have those of the brain, though many of the remarks we have made upon the latter will equally apply here. Thus, in idiopathic inflammation of the membranes, the exudation is from the pia mater, and consequently beneath the visceral arachnoid, as in the brain; only in very acute cases of idiopathic inflammation may a little lymph be found on the free surface of the arachnoid. But you will please notice that the dura arachnoid of the spinal canal is not quite the same in its relations as the dura mater of the cranium, for it does not serve for periosteum. It is, indeed, a special membrane of the cord. Also notice that the arachnoid is much more distinct from the pia-mater, and the subarachnoid space much more defined and regular in the spine than in the cranium. We will again consider these parts as dura-arachnoid with arachnoid cavity, and pia-arachnoid with subarachnoid space.

DURA-ARACHNOID AND ARACHNOID CAVITY

Acute Inflammation.—This is rare in the dura-arachnoid. The membrane may be involved in an acute process in those cases where disease has penetrated from without. Thus in caries of the spine, the sheath of the cord may become involved in the inflammatory processes, and the same sometimes occurs through necrosis of the sacrum, arising from bed sores, and extending to the meninges in the spinal canal.

Let us take an example, the case of a lad who had received a blow over the loins; an abscess formed which involved the vertebræ, and the purulent matter entered the spinal canal, so that when he died, three weeks afterwards, the arachnoid cavity was filled with pus, and the inflammatory process had proceeded up to the brain, and there produced also an arachnitis. Several similar cases have occurred from bedsore, where, owing to necrosis of the sacrum, the lower part of the spinal canal was laid open, a purulent inflammation extended along the arachnoid, and also reached the skull. Thus, a man had paraplegia for a month; bedsore formed and extended frightfully, causing necrosis of the sacrum; the inflammation then reached the meninges and set up a dura-arachnitis; the arachnoid cavity was filled with puriform lymph. In this way the original spinal disease—softening of the cord—produced indirectly a second and totally different spinal disease; we have now seen several such cases.

Chronic Inflammation.—Most of the changes seen in the dura mater are of a chronic character; thus in disease of the spine, of which we have been speaking, after the canal has been reached, some lymph or scrofulous matter is formed on the outer surface of the dura mater, and extends sometimes along it for a considerable distance, and so thick layers of deposit are found covering it; the inflammatory process may extend through the membrane, or the tuberculous matter may be found protruding on its inner side; but it is surprising how long the dura mater will resist the passage of inflammation. Unhappily, however, it does not prevent the tuberculous abscess pressing on and destroying the cord, an effect that may be produced when the curvature of the bone is very slight.

In chronic inflammation, not arising in the bone, the medulla and all the membranes are involved together, with local or general adhesions; just as we find in the brain, so in the cord, it is often difficult to say in what structure the morbid process commenced, or whether it has not proceeded in all *pari passu*. In these specimens you see the membranes adherent to the cord, and in this one the dura mater is ossified, and in some parts forms a complete bony sheath around it; in this drawing you will see how it appeared by the microscope, consisting of true osseous structure. In some cases chronic spinal meningitis is evidently syphilitic, coexisting with gummatous disease in the liver, &c.

Tubercular Meningitis.—This affects the dura-arachnoid of the cord, although it very rarely indeed, if ever, affects the cranial dura-arachnoid, and then only in traces; but in the spinal canal you very commonly, we should say in most cases of tubercular meningitis, find the dura-arachnoid to be crisp, thick, and vascular-looking, and to have its inner surface sprinkled thickly with numerous fine, sand-like tubercular grains. Naturally the spinal dura-arachnoid is very thin: if laid on fine print you can see to read through it quite plainly. I these cases of tubercular arachnitis it often becomes too opaque f this. It is now some years since we first drew attention to this curious pathological difference between the cranial and spinal dura arachnoid.

Morbid Growths.—The tumours that affect the dura arachnoid mostly proceed from without to attack this membrane. Thus tuberculous disease of the spine, or cancer beginning either in the bones or around the spine, penetrates to the canal, and involves the sheath, or even the cord within. Many specimens of this you may see on our shelves. In one curious and interesting case observed by Mr Campbell De Morgan, a cancer in the anterior part of the base of the skull was associated with cancer in the meninges of the spine, suggesting that the germs of cancer had spread along the sub-arachnoid space.

Virchow gives a remarkable case of enchondro-lipoma, which arose in an old soldier, between the bones and dura mater in the dorsal region, and compressed the cord fatally. He thought it might be congenital. Somewhat similar are certain compound mucous and cartilaginous tumours. Thus, we found a tumour congenitally developed in the sacral region attached to the meninges at the lower end of the spinal canal; in this there were cartilage, bone, brain, ciliated mucous membrane, &c., in short, almost all the tissues of a fœtus. Such tumours are more frequent in the sacral and coccygeal region; they are generally situated in the middle line. They appear to represent partial budding or division in early fœtal life, or "included ovum," &c. Tumours arising primarily within the compass of the spinal dura mater, or from its substance, are really rare. The few that have been recorded are generally spindle-cell and fibrous growths, which come from the arachnoid face of the dura arachnoid in the same way as similar growths spring from the arachnoid side of the dura mater of the skull; but they generally, in the spinal canal, prove fatal before they have reached any size. Those which we have seen have generally been about the size of a hazel nut, and of an oval shape; by pressure on the medulla they have destroyed its texture, and produced death by paralysis in the usual way. A remarkable case recorded by Dr Cayley of a tumour with sandy grains encysted in a fibrous tissue evidently corresponds with Virchow's psammoma.

Cancerous tumours have, we believe, been occasionally met with. We have never seen them primarily spring up here, but growths may, as we have said, proceed from without, and destroy the membranes.

It is important to notice that in those cases of multiple round cancer between the cranial dura mater and the bone, there are often numerous cancers in the bones of the spine, but none on the dura mater there; proving that in the cranium they belong to the bone.

PIA-ARACHNOID MEMBRANE AND SUBARACHNOID SPACE

We take these together because pathologically they suffer together like the corresponding parts upon the brain.

Inflammation. *Acute.*—The superficial or arachnoid membrane is almost evascular, while the blood-vessels are in the deeper membrane lying immediately over the cord; consequently all idiopathic inflammatory exudations take place from the deeper membrane, and are collected beneath the arachnoid. This, you see, is analogous to what we told you with respect to inflammation of the membranes of the brain, that in this affection the exudation in primary inflammation was not interarachnoid, or between the serous surfaces, as you might have thought by judging of the analogous instances in the other serous cavities of the body, but beneath the serous surface in the subarachnoid space. You must remember, however, that in the spinal as in the cranial membranes a pia-arachnitis may arise from external causes, though a dura-arachnitis does not arise from internal causes. Spontaneous spinal meningitis therefore is the same as pia-arachnitis, and we look for the products of inflammation under the arachnoid surface. What first strikes the eye before opening the membrane is the remarkably irregular look of the cord, owing to the effusion of lymph within the arachnoid space; this space, you know, can be distended with air into a large sac, and being now occupied irregularly by inflammatory exudation, the whole surface of the medulla acquires a very uneven aspect. On cutting the arachnoid the lymph is found beneath as a yellowish-green material, toughish, softer, or even puriform; in some cases it is more in the front, and in others on the posterior part of the cord.

It is not often that you meet with acute spinal pia-arachnitis without the cerebral membranes being affected at the same time; you know how freely the cerebral and spinal arachnoid spaces are continuous.

Cerebro-spinal meningitis; cerebro-spinal fever.—Acute meningitis of the cord is generally speedily fatal. This of itself would ensure attention to it, but its most serious interest is in respect of its occurrence in the form of the so-called cerebro-spinal fever, which has been epidemic on the Continent, in Dublin, in America, &c., though never to any extent in our own island. The nosological relations of this disease are yet uncertain, since Dr Murchison has leaned the weight of his authority in favour of a doubt whether the arachnitis is more than a complication of typhus, &c.; standing to this, he suggests, in the same relation as miliaria has stood to fevers in some Italian and other epidemics. But whatever its nosological nature there is no doubt that this meningitis gives a grave and fatal character to some epidemics. "Cerebro-spinal meningitis" is another common name for it. Like other grave fevers it may come down on its victim altogether overwhelmingly, so that he becomes dusky and purpuric, and sinks in a very few days before any very obvious products are formed in the meninges. But when the case is as usual of mitigated yet fatal severity you find it a pia-arachnitis, the lymph lying chiefly on the dorsal surface of the pia mater. Only in the very rarest cases do any products appear in the arachnoid space. The spinal cord itself in this disease is often quite free from change, but sometimes a slight inflammatory formation is traceable along the lines of the medullary rays for some little distance into the medulla.

Our own experience of pia-arachnitis or spontaneous meningitis is limited. We have met with it as a complication of the late stage of heart disease. Dr Murchison has recorded a case of the same kind. We have twice found it in persons with evident constitutional syphilis, once in a woman with syphilitic rupia and characteristic gumma of the liver; once also in pyæmia. One thing we would remark here, though the point is rather clinical than pathological, and that is, that the set of symptoms which even in our latest text books are given as characteristic signs of spinal arachnitis, viz. tetanoid stiffness and pain in movements of the limbs have not existed in those cases where we have verified the meningitis post mortem. Thus, in one case a boy who had pus filling up his subarachnoid space stood up in his bed restlessly, and got out to micturate only a few hours before his death. In another case the woman was found restlessly wandering from bed to bed on the only night she lived after admission to the hospital. In another the limbs were described as "tossed about." Another suffered from languor, heat, thirst, and, finally, convulsions. For instance, the description of tetanoid state in Dr Reynolds's 'System of Medicine' corresponds more to tetanus; and as in the case given as typical there were no post-mortem signs of inflammation, it can scarcely be considered certain that the case was not one of tetanus.

Chronic inflammation.—The pia-arachnoid is occasionally found thickened and opaque in persons who have long suffered spinal symptoms. It then is commonly more or less adherent to the dura mater. This appearance, on the whole, is very rare. Most of the chronic spinal diseases implicate the cord proper and not its membranes. It is necessary to note that there are fine adhesions by minute points between the dura- and pia-arachnoid normally present in the cervical region. These we have often found incorrectly described as a pathological state.

Local gummatous *syphilitic* inflammation has very rarely occurred. It resembles exactly the like gummatous meningitis of the brain.

Congestion and Apoplexy.—We have never seen a case of apoplexy of the spinal membranes, but such disease occasionally occurs, and gives rise to sudden paraplegia. Blood is frequently found in the spinal membranes as an extension from intracranial hæmorrhages, either traumatic or spontaneous. We have known it produce additional symptoms in such cases. As to congestion we would refer you to the remarks we made on congestion of the brain. Post-mortem fulness of blood can never prove an active state of congestion, nor have any real pathological importance. Owing to the mode of death, and the body having been long supine, the veins may be found filled with dark blood. Perhaps the chief use of congestion of the membranes, after death, is to tell you quickly which is the posterior and which the anterior surface of the cord after its removal. For the blood here, as in other parts, gravitates to the back of the cord as the body lies. The vessels, too, are here more tortuous and larger. In tetanus, &c., you often meet, in the report of inspection, with the statement, *membranes congested;* this is very likely to be true, but is utterly unimportant. Neither is it very constant in tetanus.

Morbid Growths.—Tumours on the pia-arachnoid of the cord are very rare; we have but one specimen composed of wavy fibrous tissue and long nuclei. It is said that *carcinoma* has occurred there, and Virchow remarks that *sarcoma* is more frequent on the spinal than on the cerebral pia mater. Once we met this curious elongated *cyst*, three inches long by half an inch wide at its widest part, on the dorsal aspect of the lumbar part of the cord, firmly connected with the free surface of the arachnoid, reminding of the hæmorrhagic cysts in the cranium, but the latter are more connected with the dura mater.

The spinal arachnoid, however, very commonly is the seat of curious *bony* plates. You remember that these were very rarely met with on the pia-arachnoid of the brain. They are so common over the cord that often we have found them there in each of five or six bodies successively; half the population at thirty-five have a few. It is a great and a rather common proof of ignorance of the spinal cord to treat them in a post-mortem examination as if they bore on the symptoms of the case.

They are white, smooth, irregular-shaped plates on the arachnoid seen on opening the dura mater. Their most frequent seat is at the lower and posterior part, especially on the cauda equina. They are white, like porcelain, or, when very small, like grains of split rice. If you try to remove them, they are found firmly united to the membrane all around their circumference; and you will notice that though the outer face is smooth the inner is surprising from its extreme roughness due to papillose projections, highest at the centre of the patch. These have long been known by the name of bony plates on account of their hardness; but the correctness of the name has been questioned, because often no bone can be found in them, and they have therefore been called cartilaginous. The tissue of which they are composed is very like bone, but often without lime-salts. You see in it cells with out-branching processes, resembling bone-cells, with more or less of hyaline or fibrillated intervening substance. The branches of the cells often meet, so that when calcification sets in the transformation to bone, with its lacunæ and canaliculi, is immediate; the result, not being, perhaps, perfectly typical bone, yet sufficiently characterised.

As to the significance of these, they were once thought to cause epilepsy, tetanus, &c.; but they are probably innocent, at least, we have often found them largely developed, without any spinal symptoms. Virchow suggests that they rather result from than cause irritation. But the freedom from symptoms is surprising when their size and number, and their roughness towards the cord is considered; we counted thirty-four large plates in one case. They are rarely on the front of the cord. They increase with the age of the individual, but we have found many large ones in a person of twenty-four years.

MEDULLA SPINALIS

Inflammation.—Acute inflammation of the spinal cord is rare. It is described as sometimes producing pus. We have never met with this occurrence. Analogy with the brain would suggest that spontaneous suppuration of the spinal cord would be most rare. The brain only suppurates from grave injuries or pyæmia. The same is probably true of the cord. It is remarkable that one cannot meet any detailed accounts of such suppuration given by competent observers. The nearest approach to pus that we have met was in the case of a girl who was ill two weeks with total paraplegia, that had completed itself in a quarter of an hour from the first moment of the attack. The central grey matter of this cord was swollen, and in many places sulphur coloured. The microscope showed besides disintegrating nervous elements, a vast number of well-formed spinal cells all diagrammatically like the elongated fibre cell of inflammatory lymph. This state was almost limited to the grey matter, and when affecting the white had obviously spread from the grey. This matter, however, was far from being pus.

Acute red softening.—When speaking of subinflammation of the brain, we mentioned some evidence that degrees of inflammation similar to those of catarrh, &c., affect the nervous tissue without forming any product in it. The negative results of some acute spinal cases may be referable to such inflammation; good microscopic examinations of hardened sections of such cords are much wanted. The cases are rare.

White softening.—In many cases of rapid death from acute spinal disease, the cord is found a little softened and pinkish; its fibres disintegrating more or less evidently, but with no evidence of formed products and no sign at all of inflammation of the membranes. The nature of such cases is open to question. We would lay stress on the fact that white softening of the brain often causes absolutely sudden, and total paraplegia in spite of the chronicity in its pathology. Again we have met examples of white softening of the lower end of the cord, which although they lasted months, yet they had produced total paralysis quite suddenly. Thus it appears that the cause of white softening is apt to quickly paralyse the part affected, and it is only if life be continued, that the part goes through a gradual degeneration, producing the granule corpuscles characteristic of white softening. Now, if this cause of white softening attack the cervical cord, death ensues too swiftly for the change to acquire its histological characteristics, and then you would, probably, meet with no evident morbid anatomy in the cord.

When white softening affects the lower end of the cord, the characters assumed by the affected part are like those of white softening of the brain which we have already fully described (p. 235); you rarely, however, find the cord completely diffluent like the brain becomes, and we must get you to remember this curious fact, that you meet with cases where the granule masses in the cord, and the disintegration of its fibres, such as is described in softening of the brain, are quite characteristic, and yet there is no loss of consistence. These cases of white softening of the lower end of the cord are clinically "*chronic paraplegia*" of adults. Further observations on the pathology of this, especially its relation to the grey and white matter of the cord, are much wanted. We will shortly warn you of two fallacies in inspecting such cases; remember that in the common process of removal of the cord it is very apt to receive a blow from the bone forceps, and so exhibit a very decided and unquestionably local softening, and also remember that when the cord is swollen as well as soft its membranes are tense, and the cord though very soft may appear morbidly hard. But on section of such cords the soft medulla bulges out, and the action of water reveals its softness. These sources of fallacy are most important yet too often ignored. They make the ordinary accounts of a softened or hard cord really worthless.

Induration—Sclerosis and Grey degeneration.—We have already spoken of *insular sclerosis* (sclérose en plaques) of the brain. This condition with exactly the same morbid appearance (see p. 230) is met with in patches throughout the spinal cord both in the grey and white matter. It is occasionally found limited to the cord, indeed more frequently than it is limited to the brain. This condition in the cord was, we think, first described by Charcot, as grey degeneration of the lateral columns in a hysterical subject; we mention this because such cases are often still called hysterical disease, though the peculiar jactitating, trembling, and stiffness, &c., give early notice of its real nature.

Grey degeneration is found in the cord in bands extending through great lengths of its tissues; this is especially the case in the posterior columns where it gives rise to *locomotor ataxia.* From its distribution this sclerosis is called "*ribbon like*" (rubanée), in contrast with the *insular* sclerosis. Its chief importance is in locomotor ataxia; it usually implicates not only the posterior columns, but the posterior roots of the nerves and the neighbouring parts of the lateral columns and grey matter; the meninges may be rather thick over the part. The microscopic appearances show removal of the oily medulla from the nerve fibrils, and some augmentation of the connective rays in the neighbourhood affected; the nerve fibrils thus waste, and their place is taken by increased connective substance in which amyloid corpuscles abound. In our specimens the capillary vessels persist, though we have heard this denied; the central axes of the nerve fibrils are found very long after the oily matter has disappeared. The outline of these nerve fibrils in transverse sections is still confusedly shown by the thickened neuroglia surrounding each central axis with a slight interval; we have traced them still persistent in the oldest parts of our specimens, showing that the change is gradual and delicate, by no means to be thrown into the class of mere rough inflammations. Discussion as to the nature of the change has been almost violent. Prof. Leyden sums up the evidence against the opinion that it is due to meningitis thus: 1. The signs of chronic meningitis over it are not constant, grey degeneration may exist without any meningeal disease. 2. The degeneration he has found in early cases not to attack the surface first. 3. Hyperæmia of the vessels means nothing since the tissue is succulent and soft. 4. The pain is probably neuralgic; and, 5. Other meningitis does not lead to the result.

The distribution of the change along the tracts of the nerve columns in locomotor ataxia proves that the change belongs to the nerve and not to the neuroglia. Yet the neuroglia is changed; it is perhaps like what occurs in the burning of coal and clay to make ballast; the coal burns away, and the clay remains dense and hard. In the sclerosis of ataxia, the nervous tubules are destroyed by some active process which we may call by our coarse term inflammation; they burn away, as it were, consuming themselves in their irritation, for it is not their nature to resist passively and thicken. But the neuroglia remains and is thickened, because its only way of showing irritation is to thicken.

Although the names "sclerosis" and "grey degeneration" are applied equally to the "insular" and "ribbon-like" form, yet they have not the same microscopic appearances. In sclerosis you meet with numerous granule masses, especially in the brain, but we have not generally found these in the grey degeneration of posterior columns, while this is much softer. We think sclerosis and grey degeneration should be distinguished as different conditions; the former originating in the neuroglia, and the latter in the proper nervous elements.

Paralysis agitans.—Few observations have been made on the cord in paralysis agitans; its condition is not known satisfactorily. One examined by Dr Cayley showed increase of the connective substance on the periphery of the organ, and in the medullary rays. This corresponds with the opinion of Charcot, that in spinal disease without wasting it is the white matter, but when with wasting it is the grey that is affected.

Infantile paralysis.—"Sclerosis," a term often used indeed for all chronic grey changes in the cord, whether they are hard or soft, is also the name given by some to the changes described in the grey matter of the cord in infantile palsy by Prevost, Charcot, Jouffroy and others. This change consists of destruction of isolated groups of ganglionic cells in the anterior horns of the grey matter, others around being unaffected. Charcot says the change begins originally as an inflammation of these cells, and spreads secondarily to parts around. Some cells recovering, while others perish entirely, so that a partial recovery of the use of the limb is often allowed.

Progressive muscular atrophy.—In this it is said the great motor cells of the anterior roots disappear. We have some specimens prepared with great skill by Dr Thompson Dickson; they bear out the statement which attributes the disease to this cause. In some sections there is scarcely a cell to be seen, yet it is curious that the whole bulk of the grey matter is not diminished. We have seen a similar grey change about the nucleus of the ninth nerve in glosso-laryngeal palsy. Whether, as Bouchard thought in such cases, the change in the cord arises by extension from the nerves which are also wasted, is a question that must be at present held to be doubtful.

The pathology of *general spinal paralysis* is not yet known satisfactorily. Dr Lockhart Clarke exhibited some specimens showing a shrivelling of the large cells of the grey matter, which were reduced to the natural size of their own nuclei.

After amputation of a limb the corresponding part of the cord shows some granular changes and wasting of the nerve cells, but no disappearance of them as in muscular atrophy; we think this shows that the latter cannot be a secondary change due to the muscular wasting. The changed state of the cord and cerebellum after amputations was especially studied by Dr Dickinson.

In leprosy.—Daniellsen and Boeck described important changes in the cord which was found by them shrunken and unequal. Dr Carter, of Bombay, in sixteen cases found it healthy; in two cases that we examined there was no disease of the cord.

As a sequela of *fractures of the spine* with crushing of the cord when the patient has survived many weeks, we have found singular changes in the cord extending several inches from the injured spot; in one of our cases the grey matter has shrunken to one-eighth of its natural size, five inches from the seat of injury, and in the shrunken grey matter only inflammatory and hæmorrhagic products are to be seen; this is interesting in connection with the loss of electro-motility, which Duchenne has shown to supervene early after fracture of the spine. Destruction of the grey matter to a distance from the injury would account for this.

Apoplexy of the Cord differs, according to Charcot, from cerebral apoplexy, in that it supervenes on inflammatory states; but like cerebral apoplexy it affects the grey matter, in which it may extend to a great length. This condition is very rare, we have ourselves never met with it.

Tetanus.—The cord in tetanus appears quite natural to the naked eye. There is generally much blood in its vessels; this is due to death by asphyxia; it is not constant. Thus we had two cases of tetanus together on the post-mortem table; in one of them the cord was congested, but the other showed one of the whitest cords we ever saw. The microscopic alterations are not satisfactory; this you might expect when you reflect upon the tetanus of strychnia poisoning, in which you may very nearly tetanize the subject to death, as in Trousseau's strychnia treatment of chorea, or in experiments on dogs; so that if you gave a very little more the case would be fatal; yet if you hold your hand, recovery is immediate. How then should you expect to find a changed anatomy in the cord, when just short of death it soon proves its soundness? Accordingly, although some observers describe a number of minute alterations, Dr Albutt, for instance, enumerates eight changes, such as congestion and escape of blood, granular degeneration, altered nerve cells, &c., others differ much from them, Drs Lockhart Clarke and Dickinson, for instance, found in Dr Albutt's specimens only three changes, and one of them was not found by Dr Albutt, while another was found only once in four cases, and they differed from his view of it, so that only in a single point did they support his view. Thus, competent observers differ much upon the points of minute detail that are described as the results of tetanus. We have made numerous examinations and have seen the vessels full of blood, and some granular changes in the nerve fibrils around, but we have found the same changes in bodies with no spinal symptoms, and we have observed the same changes in the skin of the tetanic cases. We also observed that these changes occur especially in bodies that have lain some time after death in warmer weather, and when the vessels are much engorged through the plethoric state of the person, and death by asphyxia. In some cases of tetanus they are entirely absent, and these are the cases that are examined quickly after death. Hence, we think, the granular states described by Dr Clarke and others, are widely open to the suspicion that they are of post-mortem origin. So far post-mortem evidence agrees with considerations drawn from strychnia poisoning, in asserting that tetanus, like epilepsy, nay, like violent coughing, has no morbid anatomy except, perhaps, its traumatic cause, and the asphyxial congestions resulting from it.

The morbid anatomy of the cord in *hydrophobia* is in the same position.

Morbid Growths.—*Tubercle.*—The most frequent growth in the spinal cord is a form of tubercle, which has all the characters of tubercle of the brain, forming a large mass of the colour of milk of sulphur in the tissue of the cord. In all the examples we have seen, the tubercle commenced in the grey matter of one side, and expanding in the tissue around, left only a thin superficial layer of cord substance unaffected; it is worthy of your notice that there was scarcely a trace or sign of the presence of the tubercle in the outside view of the cord, so that it would be easily overlooked unless the whole cord were sliced carefully. Its microscopic structure showed in one instance most plainly an extension of the lymphoid tubercle all along the sheaths of the vessels in the cord. Another point of interest in this case was the limitation of the tubercular disease to that zone of the body which corresponded to the tubercle in the cord. The tubercle was in the upper part of the lumbar bulb, and there was slight tubercular affection of the peritoneum of the middle of the abdomen with slight tubercular disease of the mesenteric glands. In this case death was caused by the spinal tubercle only; it is not then correct to say that tubercle of the spinal cord only occurs in cases of advanced tubercular disease. It may be primary; indeed, in this nodular form we believe it is always primary.

Syphilitic gumma has been found in three or four cases in the spinal cord; we described one of these in the 'Guy's Hospital Reports' for 1870, it had the usual characters, that is, destroyed the affected tissue, and replaced it by gummatous matter.

Tumours of ordinary character arising in the cord as distinct from the membranes we have never met with, nor do we know of any descriptions of cases, but it is stated that carcinoma or sarcoma have been found; we have seen them reach the cord from without. We have, however, seen a section of a most remarkable growth that wa formed as a swelling in the dorsal aspect of the lumbar bulb. I occurred in a woman æt. 33, under the observation of Dr Seguin, a American physician; the woman died of pressure on the cord by meningeal sarcoma in the cervical region; a section of the lumba growth was sent to Sir W. Gull, by whose kindness we saw it. Th swelling consists of a partial repetition of the cord in continuity wit its proper texture, so that there is lengthwise another smaller cor which is within the pia mater, and has its own white matter, gre horns, nerve cells, and commissures; it appeared to us to be congenital malformation.

Hydromyelocele.—Sometimes the central canal of the cord is foun dilated to a greater or less width, and at one spot or at many spots all along its spots. This state has been repeatedly found withou any spinal symptoms, as in this example which we took from a boy wh had had no nervous disorder, but in whose thorax was, curiously, a ban of thickened pleura on each side, to explain which we searched the co and found this dilatation at the point corresponding. In one ca where symptoms were present Gull found the tissue around such dilatation in a softened state; the symptoms were no doubt due the softening and not to the dilatation.

Hydatids.—Rokitansky speaks of cysticerci as having occurr repeatedly in the cord, but the acephalocyst hydatids outside. W met with a case of crushing of the cord by multilocular hydatid extending from the spine; they did not pierce the dura mater.

Injury.—Cases of recovery from injury of the spinal marrow a recorded, yet no new formation of cord tissue is known to occur, that after grave injuries paralysis is permanent. In fractures dislocations of the spine, the cord within is often frightfully injure as you may see by examining our preparations. The cord may be to through, and the ends forced more than an inch apart, being connect by lacerated membranes, or the cord may be pierced by fragments, burst, or, in many cases, the membranes are unaffected, but t mischief is caused to the medulla within by stretching and contusio whereby the structure is softened into a pulp, and is red from effus blood. Sometimes we find the outside of the cord little injured, b the interior grey substance softened; this arises from the softness a vascularity, which allows the blood to be more readily effused into and even to proceed some way downwards along its centre. We ju now (p. 252) spoke of the after effects of this extended injury. Dea occurs in these cases not so much from the amount of injury produc as from the particular spot affected; thus, the instances of recove from fractured spine are those where the lower dorsal or lumbar vertebræ are injured; where immediately fatal, the injury is high up in the neck, above the third vertebræ; and in the more usual class of cases, where death occurs in one, two, or three days, the injury is at the lower cervical or upper dorsal region, so that the ribs being paralysed, and the respiration only effected by the diaphragm, the lungs become gorged, and death sooner or later inevitably results. Possibly, where a violent blow has occurred on the neck, without any displacement of the bones, contusion of the cord may have arisen from concussion.

NERVES

Atrophy of the nerves arises from pressure, or is a mere accompaniment of a general atrophy of the part of the body which they supply, you will find the recurrent laryngeal nerve wasted and almost destroyed from the pressure of an aneurism. In cases of dislocation of the arm, the circumflex and other nerves have been injured, causing paralysis. The same may occur through the use of ill-fitting crutches. A spontaneous or *idiopathic atrophy* of the spinal nerves has been said to cause progressive muscular atrophy. This, if true, would not determine whether the muscle or the nerve is first involved.

Injury.—Experiments on animals have shown that, if a nerve be divided, new fibrillæ are formed, and a perfect union again occurs. It has even been shown by Vulpian and Phillipeaux that a portion of motor nerve may be interpolated into the course of a sensory nerve from which a piece has been removed, and after time allowed for complete union the conducting power will be re-established through the foreign cord. In the human subject, too, we are sure that where the nerves are small, the same reparative changes occur, as, for instance, in a cut finger, where sensation is for a time lost and afterwards regained. We do not, however, witness such a union, or but very rarely, if the nerve be large, and, therefore, it is only in small nerves, and when patient is young, that we can expect to meet with this result. After amputation, the ends of the nerves very often become enlarged by an effusion of fibrinous material into them, and become *bulbous*, a condition which, in order to be distinguished from a similar one we shall presently mention, is called *traumatic neuroma.* In this preparation you will see what we mean; the nerves, as they approach the end of stump, become enlarged, and this you constantly see where there has been no complaint of pain; though very often, when adherent to skin and involving the cutaneous nerves, they cause excessive distress.

Inflammation.—This is constantly spoken of in connection with various neuralgic affections, but it is to a great extent hypothetical, as it is not often demonstrated after death. On the other hand, we have once or twice found the large cords of nerves on the sciatic, surrounded on all sides by pus, without any remarkable pain having been suffered; but in one of these cases the patient died of tetanus. It is not unusual to find a nerve involved in an inflammatory process, as, for example, in a stump, where it may be seen covered with lymph, and this may, indeed, be the first stage of the bulbous condition already spoken of. You may find a nerve in this position covered with lymph or granulations, and enlarged. Such a condition of nerve has been often found in a wounded part in fatal tetanus, and has been looked upon as the exciting cause; without saying that there is no connection between them, as very probably there may be, you may find such inflamed nerves without tetanus, and tetanus without the inflamed nerve. Such an inflammation has been said to arise in the sciatic nerve, as a cause of sciatica, but we do not know that this has ever been demonstrated. In anæsthetic leprosy Virchow showed that portions of the great cutaneous nerve trunks, corresponding to the anæsthetic regions, are swollen considerably, and through the enlargement of the component fasciculi in these a quantity of small cells, likened by Virchow to the substance of granulations, separates the individual fibrils, which meantime waste away. These observations are confirmed by Dr Carter, of Bombay. A very similar change is described by Charcot as occurring in old contracted paralysed limbs.

Morbid Growths.—The most marked changes in the nerves are of the character of tumour; such a disease of a nerve is called *neuroma*, whatever be its nature. The term chronic inflammation, however, might be employed in some examples, as in this specimen. These nerves are from the upper and lower extremities of a woman who died lately in the hospital, and you will see that nearly every one is enlarged by an adventitious deposit; this consists of a simple fibre tissue, infiltrated among the fibrillæ, so that they are firmly united together and with difficulty separated; in some portions a large mass is connected with a fasciculus, so that a tumour is produced, forming a nodule on the nerve, the other fasciculi passing over it. In other parts, the fibre tissue is diffused amongst the nerves, so that, on dissection, they are merely found separated by the new material. One of these, you see, is of the size of an egg. These tumours are of very slow growth; Mr T. Smith showed some fibrous neuromata which had been forty years in formation. In this instance, the pneumogastric nerves had similar tumours upon them. The complaint is not a painful one, and must not be confounded with painful subcutaneous tumours, consisting of nodules of fibre tissue containing fibrillæ of sensitive nerves. Sometimes, one nerve may be affected, and if this belong to an important part, like some intracranial nerves, may lead to death. There is reason to believe that neuroma, in many instances, has a syphilitic origin, the adventitious material being of the same kind as that which forms in syphilitic nodes, &c. Occasionally the tumour softens in its interior, and a cyst may be found within; into this hæmorrhage may occur, and thus several examples of blood cysts on the brachial nerves have been recorded. We have met with destruction of the facial nerve in the canal of Fallopius by effusion of blood into it.

The nerves sometimes show tumours composed of nerve-fibres and cells. Virchow calls these true neuromata; we have never seen examples. One very remarkable case is related of a large tumour in the situation of a sympathetic thoracic ganglion, composed of nervous tissue. They more frequently occur on the nerves of the limbs.

Some examples of myxoma of the nerves have been recorded, and some of these have proved malignant in their nature. Myxoma appears to be the most frequent of the single tumours of nerves, but some cases of sarcoma are recorded.

The various forms of cancer often destroy nerves in their neighbourhood, but do not often spread in their tissue. In this specimen, however, you may see the nerves of the brachial plexus enlarged by cancerous deposits, which also continue along their branches. The invasion of nerve roots along the spine in cancer of the vertebræ is apt to produce a most painful form of paraplegia.

DISEASES OF THE ORGANS OF SPECIAL SENSE

The proper diseases of any of the natural tissues, such as bone, or fibre, or vessel, exhibit a coarse disregard of the delicacy of figure in which such bony, fibrous, or other parts are formed to discharge important special offices. We have already seen how apoplexy invades the pons as unscrupulously as gout, the prime minister; one might say it is a pathological law of the economy, that if bone or fibre is used in any structure, however important and delicate, such as the ear or eye, the morbid liabilities of bone must be undertaken along with its advantages. There is one way in which the design of the body obviates the risks common to ordinary tissues, and that is by calling into existence a peculiar tissue for a vitally essential purpose; such a tissue as cornea or lens; but this is a plan little used. In the most delicate of our organs we see that the constructive work is, for the most part, done with substances which have the anatomical nature and pathological propensities of constructive elements, such as are commonly found in similar use in other parts of the body.

We make these remarks in regard of the organs of special sense because such views form the key with which morbid anatomy simplifies the problems that the complex clinical phenomena of the eye and ear place before the surgeon.

EYE

In the eye the several coats are composed of structures which have diseases like to the diseases of the same structures elsewhere; thus the diseases of the conjunctiva are those of mucous membranes; the diseases of the sclerotic are those of fibrous tissue; the diseases of the choroid are those of the pigmented membranes of the brain; the diseases of the retina are the diseases of brain, of which the retina is originally a bud.

Hence it is convenient to consider the diseases of the eye as to the coats they affect; we can only give you a cursory summary of them, and refer you for further details to Mr Bader's admirable book.

Conjunctiva.—In this we meet with the usual inflammations of mucous membranes. Indeed, these inflammations are, perhaps, best studied in the conjunctiva where their varieties and peculiarities are most easily recognised. You often see a simple *catarrhal* conjunctivitis or "ophthalmia" through exposure to cold or other irritation; in this there are only physiological or functional changes, including great active congestion, and production of mucus rendered watery by the increased secretion of the lachrymal gland. *Purulent* ophthalmia is rarer and graver, in it the epithelial cells have outwandered pus cells between and among them; the mucous membrane itself is greatly swollen and may be full of pus cells, and the cornea, iris, &c. may be attacked so that sight is destroyed; a variety of this with *diphtheritic* membranous exudation is described.

Herpetic conjunctivitis appears in the form of little red-based elevations near the margin of the cornea or the sclerotic; *strumous*, with specks on the cornea and great irritability of the eye; and *granular* where after long continued irritation a papillose condition arises on the mucous surface of the lids. *Pterygium* is the name given to triangular shaped vascular thickenings, which extend from the margin of the cornea, expanding outward, generally towards the semilunar folds.

The conjunctiva is subject to *pigment* patches such as are natural in the dark races, little yellowish collections of *fat* may form under it in the exposed part in elderly people. *Nævus* is found on it, also small *dermoid tumours* furnished with hair; *epithelioma* and *melanotic* cancer are also found in it, and *cysticerci* have been met with.

Cornea.—Although the cornea is a peculiar tissue remarkable for its richness in nerves, yet its general characters ally it closely with the fibrous textures; it is liable to a senile fatty degeneration which appears as a whitish opacity of the circumferential unused portion, generally under the upper lid; this may extend all round the eye; it is known as *arcus senilis.* The cornea undergoes several forms of *inflammation. Syphilitic* corneitis appears first as pale grey flecks which spread and unite to form a general subopacity like that of ground glass. *Strumous* corneitis shows pale spots, without the nodular elevations that characterise *pustular* corneitis, in which pus forms between the layers more superficially or more deeply; the little abscess bursts outwards, leaving an ulcer, or inwards discharging the pus into the anterior chamber where its presence constitutes *hypopion. Ulcers* of the cornea arise either from such abscesses or originally by excavation of the tissue through pressure of a hair or other injury. If the ulcer is slight the clearness of the cornea may be quite restored, but if severe there remain opacities which if faint are called *nebulæ*, but if opaque and white are called *leukoma.* At the time when the ulcer extends deeply and exposes the deepest layer of the cornea this may bulge through the superficial opening, producing a *keratocele;* or the ulcer may quite pierce through, leading to discharge of the aqueous humour, when the iris is apt to fall against and unite with the cornea—*synechia;* or bulge through the hole, at the same time being covered with lymph—*staphyloma.*

Sclerotic.—The sclerotic, like ordinary fibrous tissue, is liable to *syphilitic and rheumatic inflammation.* You also meet with *ulceration* of its surface, but much more rarely than in the cornea. In either situation the ulcer properly belongs originally to the conjunctiva.

Lens.—The lens is a peculiar substance, evascular, and hence not liable to active inflammatory exudations. Its changes are chiefly *passive;* its substance is liable to become opaque, and at the same time changed in consistence in constituting *cataract;* it may be *fluid,* and the colour whitish grey; or it may be *soft;* or else *gelatinous,* in this state resembling the natural consistence of the lens; or it may be *harder* than natural, and yellowish. The last form belongs generally to age, the former to the cataracts of younger people. Cataract is sometimes *congenital;* sometimes it is due to *injury.*

Chambers.—The chambers of the eye may contain *blood* or *pus* or *hydatids.* Pus in the anterior chamber is known as hypopion.

Iris.—The iris and choroid are sometimes congenitally devoid of pigment—*albinism.* The iris is liable to inflammation—*iritis.* This is *simple* or *syphilitic,* or due to extension of or *sympathy* with other ocular diseases. Syphilitic iritis in its earlier stages is found often to be localized on particular parts, but it is doubtful whether it has any sufficient distinctive characters. Some small syphilitic gummata have been met with in the iris. Dilatation of the pupil is called *mydriasis;* contraction of it is named *myosis.* When the iris becomes adherent to the cornea or capsule of the lens the state is called *synechia, anterior* or *posterior.* Little pigmentary tumours, or *melanoma,* are found on the iris, some congenital, by V. Grafe; also *cysticerci* have been found in it. In short, it may have the same tumours as the choroid.

Choroid.—The choroid and ciliary muscle are liable to inflammation, a sub-inflammation of the choroid is supposed to cause *glaucoma,* in which disease the eyeball becomes hard, causing pressure through tension within it, which pressure paralyses the retina. The cause of the increase of fluid which produces the tension is not so clear as its effects, which are greatly relieved by iridectomy. The choroid may be *displaced* by tumours or effusions of blood outside it. *Bone* may form on its inner surface, between it and the retina, especially about the optic nerve. We have several times seen the *miliary tubercles* described as forming in its texture in cases of general tubercular meningitis, an association very interesting through the correspondence between the choroid and pia mater. Close beneath the pigmented epithelium, numerous *colloid* or *amyloid corpuscles* are sometimes found. These may *calcify;* this reminds you of the similar colloid, and sandy corpuscles of the pia mater and choroid plexuses.

The choroid is the usual starting-point of *pigmented tumours* of the eyeball. Sometimes these begin in the orbit and invade the sclerotic, but they generally arise from the choroid membrane, and near the entrance of the optic nerve. They grow forward at the expense of the *vitreous* till they fill the eyeball and burst through about the cornea. They are generally of *spindle-cell sarcoma* structure, rarely of *carcinomatous,* generally deeply pigmented; sometimes only partially so; Mr Hulke and others have described unpigmented tumours from the choroid. The *melanotic* eye-tumours are never secondary. They are very infectious, extending along the optic nerve locally, and starting melanotic cancer in the liver and other remote parts, often very generally.

Retina.—The retina may be *displaced* by injuries or by blood or exudative fluid or tumours between it and the choroid or sclerotic. The changes it undergoes when *in sitû* may be admirably well traced with the ophthalmoscope. It is liable to *inflammation,* which may be of several degrees of severity and varieties of effect. Thus the retina may *suppurate* through injury, or large patches of it may be *grey and swollen* from similar causes; or in milder inflammations it may appear *hazy* and greyish or white; while in some such examples *hæmorrhage* occurs in its turbid tissue, or yellowish or rust-coloured *spots* appear in it around the yellow spot and optic disc. These milder forms occur in syphilis, diabetes, or albuminuria. The hæmorrhagic and spotted forms more frequently in granular kidney. The arteria centralis retinæ may suffer *embolism,* when the arterial vessels are found empty and the veins fuller than natural. Retinal *apoplexy* or effusion of blood into the retina is caused through obstruction to the passage of blood by pressure within or tumours outside the eyeball, or it happens in inflammation, as we have already said.

Tumours sometimes arise from the retina; of these some are more slow in their formation and approximate much to mere inflammatory thickenings. Other slow local thickenings become caseous in parts, and have been described as *scrofulous* deposits; but Virchow regards these as well as a large part of the more rapidly growing brain-like tumours of the retina, as *glioma.* He says they arise from the granular layer of the retina, which he agrees with Max Schulze in regarding as, for the most part, of the same nature as the neuroglia or connective substance of the brain. Some of the rapidly growing tumours approach *sarcoma* in their characters—that is, the cells are no longer small, with small nuclei, as in the granular layer of the retina; but they are larger than mucous corpuscles, and their nuclei enlarge and become multiple.

EAR

The anatomical side of ear disease is also based on the normal anatomy of the ear.

Auricle.—Hæmatoma Auris.—The auricle suffers diseases the same as those of skin and subcutaneous tissue generally. But great curiosity and interest attach to the inflammation of the auricular cartilages and perichondrium, with more or less extensive effusion of blood, which is not infrequent in the insane, and is called *Hæmatoma auris.* There is little reason to doubt that this arises from injury; thus, cases have occurred in football scrimmages, and it is said that the change in question is perceivable on the ears in some ancient sculptures representing Roman athletes who used the cestus. In this affection the perichondrium is red with congestion, and the cartilage soft, while in places, between the two, is a quantity of thin venous blood, which may be let out. After this the ear slowly heals, shrivelling at the same time. In old cases bony deposits have been found.

The perichondrium and subcutaneous tissue of the auricle often show a deposit of urate of soda in gouty people. This is worthy of your notice, for it may be useful in diagnosis; such deposits may occur without pain. In these specimens you see tumours which grew in the lobes of ears as a reaction against the irritation of earrings.

Meatus.—The meatus likewise suffers the ordinary diseases of the skin and subcutaneous tissue. It is liable to the occurrence of small circumscribed *abscesses*, which may implicate the periosteum, and produce ulceration and necrosis of the bone beneath, *Polypoid fibrous growths* and *sebaceous* cysts also occur there, as you see in these specimens. The ceruminous glands in the deeper part of the meatus are apt to secrete an excess of ill-conditioned *cerumen* blocking up the passage. The removal of these constitutes the most effectual triumph of aural surgery.

The tympanic membrane is liable to inflammation and ulceration, leading perhaps to perforation. From its surface polypoid growths are apt to sprout; they are of the nature of large flabby vegetations.

The middle ear has a mucous lining continuous through the Eustachian tube with that of the nasal pharynx, and the inflammations it undergoes are those usual in mucous membranes. These are described as dry and moist catarrh, and thickening of the mucous membrane itself. Little is known of these states anatomically; they are mostly inferred from the behaviour of the Eustachian tube during catheterism. Catarrh of the middle ear is supposed to be the cause of the deafness that often accompanies continued fever. The inflammation sometimes reaches the bone and causes caries or ulceration of it, with production of very painful and sensitive spongy granulations, which may present themselves at the bottom of the meatus when the tympanic membrane is perforated; these are of very bad import. By a more chronic action the ossicles are liable to be thickened or ankylosed either by bone or fibrous tissue. The incus may be ankylosed to the fenestra ovalis.

In the *internal ear* few changes have been noted. The otoconia or ear powder in the labyrinth has been found in excess by Mr Toynbee. Acute inflammation, extending from the middle ear to the labyrinth, leads to acute necrosis of the bone around, which may extend to the dura mater or its sinuses, producing acute meningitis or abscess of the brain (see p. 233). A more chronic form of inflammation of the labyrinth is admitted, but its anatomical results are undecided. Thus, the changes as yet recognised in the ear are few and coarse compared with those recognised in the eye.

The *acoustic nerve* has an evil precedence among the nerves of the higher senses in its liability to neuromatous swellings. Many of these in the form of fibrous, mucous, and once or twice fatty tumours have been recorded as causes of deafness and worse.

NOSE

Here also we find the cutaneous covering and mucous lining of the organ liable to the ordinary affections of skin and mucous membrane. In glanders pustules form within the nostrils; they break and leave ulcers in the swollen membrane. We must notice in the nose a tendency to obstinate ulceration of the mucous membrane in weakly people; this occurs in scrofulous persons, and is called *ozæna*. In some of these cases the secretion forms concrete masses, which the patients bring and show you. The bone may become affected; but generally, when this is the case, the cause is syphilis, which is, anatomically, very difficult to distinguish from scrofulous disease. Syphilitic ulcers are found in the septum or the inner surfaces of the alæ nasi, in the form of roundish fungous ulcers, with yellowish crusts. If these are neglected the bone or cartilage is attacked, and comes away in smaller or larger fragments. There is danger of great disfigurement through falling in of the septum, or destruction of the cartilages of the alæ. *Polypi* of the nose, called mucous polypi, are frequently removed by the surgeon; they are soft, gelatinous-looking, and consist of a delicate fibro-cellular structure, composed of long filaments connected with stellate cells, forming a lax meshwork, with a thin mucous fluid soaking it. They are highly vascular, and the surface is covered with ciliated epithelium; so that it forms a very beautiful object for the microscope; and, on the next occasion when a polypus is removed, if you snip off a piece of the surface and place it beneath the microscope, you will see nucleated columnar epithelial cells, with their edge furnished with cilia in constant vibratile action; you must place it in a little of its own juice, for if you put water you will stop the motion. The growths from the mucous surface are not all of this innocent nature; you may meet polypi which are cancerous. These are generally sarcomata attached by a broad base, and are connected with the periosteum or bone, cranial or spinal.

The sinuses in communication with the nasal passages sometimes have their orifices obstructed, while they become the seat of inflammatory mucous flux, so that the cavity may be distended and cause great suffering. In some cases the contents occasionally burst through the obstruction, producing periodical discharges with temporary relief. Sometimes the retained secretion bulges the frontal sinuses either forwards chiefly, or towards the orbit. In considering such cases you must remember the small congenital encephalocele at the root of the nose. Sometimes round worms or insect larvæ find their way into these sinuses.

DISEASES OF THE SKIN.

We shall consider these as corresponding to the several components of the skin as far as this is practicable, dividing them into diseases of the epidermis, corium, and appendages.

EPIDERMIS

Atrophy.—An atrophic state of the epidermis together with an imperfect formation of its elements imparts to the skin a disagreeable thin dry appearance, while the surface is chaffy, or brawny, or greasy and lustrous. You see this state most evidently in old age, or in syphilitic subjects, especially infantile syphilis, and to a less degree in hepatic and other cachexias, in all which states the epidermic appendages —hair and nails—suffer likewise in their nutrition, becoming thin, falling off, or losing their colour.

Hypertrophy.—The epidermis, like all parts composed of cells, quickly increases from over-activity in its vascular supply, so that a degree of thickening of it forms a part of a great number of diseases. Thus during the superficial bright-red blush of scarlatina new cuticle is formed, which throws off the old in subsequent desquamation. The deeper seated congestions of larger vessels in measles, &c., usually fail to do so; so also often during and after intense sweating in rheumatic, scarlet, or typhus fevers, excess of cuticle is found to displace the old, inducing partial desquamation.

But besides these desquamations revealing the results of active congestion of the corium or thickening of the epithelium is found to constitute the anatomy of many forms of cutaneous disease. These may be diffused over a large part of the cutaneous surface, arising from a general cause, or involve only a small part, from the cause being local.

The diffused kinds are *psoriasis*, *lichen*, and *pityriasis* (an improved nomenclature making lepra apply, as in former times to leprosy only). The papules of lichen consist of epidermic cells heaped up in and around the hair sacs, and mixed with sebaceous matter; such papules may be detached without bleeding, but when the lichenous affection is severe it may inflame the corium, and throw the whole epidermis into a morbid state, while the hair sacs are especially diseased, and the roots of the hairs deformed. This more severe *lichen* constitutes a rare and dangerous disease, called *Lichen ruber* by Hebra. Lepra and psoriasis are varieties of the same disease. *Psoriasis* is a morbid condition showing itself in excessive formations of cuticle, whereby patches of scales are produced; and, according to the amount and shape of the patches of disease so produced, various trivial epithets are given to the affection. But in our *post-mortem* examinations of such patches we have, like other observers, not been able to find any morbid appearances, except the scales themselves, composed of compacted epidermis. The red patches of the cutis, on which they are borne during life, become pale after death, and do not differ in consistence and appearance from the cutis around, either microscopically or with the unaided eye. As to pityriasis, we shall here only remind you of that formidable disease *Pityriasis rubra*, described by Hebra, in which a universal redness of the skin, with a slight formation of minute epidermic scales, make the only anatomical sign of a disease that is almost uniformly fatal.

When the epidermis is congenitally thick and rough, the name *icthyosis*, or fish skin, is used; some remarkable examples of this you will see in our wax models and drawings, and also in this piece of skin removed from a patient so affected. The cuticle projects in little papillose processes, which, lying side by side, produce an appearance like the pile of velvet; or, becoming thicker in some places, grow so large that they resemble warts. In less degrees, there is merely a thickened scaly state of the epithelium.

Of *local* hypertrophy of the cuticle we see examples in *callosities* and *corns* arising from pressure. The former are met with on the palms of the hands of those who work hard, as watermen, &c.; thick layers of epidermis being thus produced. Corns are mostly seen on the toes, and result from tight or ill-fitting boots, but they may form on any projecting parts; they consist of accumulated masses of epithelial scales, which lie superimposed, and rise up one over the other until they form a column, whose lower part sinks down upon and into the true skin. You also see remarkable *horny growths*, composed of a collection of epithelial scales; these do not rise, as far as we know, from pressure, but from some other local irritation; a vast production of diseased epithelium takes place, and rises up by continued additions from beneath, until, in time, long spiral projections are produced, resembling horns, or like the chemical toys you may have seen under the name of Pharaoh's serpents, which grow more rapidly, but in a very similar manner. If examined, they are found to be composed of epidermic scales, massed together in layers; there is also amongst them a little fatty or sebaceous matter, but we do not know that they always have their origin in sebaceous follicles. This specimen of a horny growth came from an old woman, who, about twenty years before, noticed some scabs on her head and body, and these gradually increased in elevation during some years, until three horns appeared. These spontaneously dropped off; but they all began to grow again, and then a second time fell off; and this is one of the last crop. Here is another smaller one, which Mr Cock removed from a man's lip. These must not be confounded with very similar looking horny warts of epithelial cancer.

Alterations in Colour.—These alterations in the skin chiefly depend upon changes in the epidermis, though sometimes a deposit of pigment occurs in the corium; and the colour of the skin is influenced also by the state of vascular supply in the corium, by effusion of blood or petechiæ, by fatty degeneration, &c. All these simple alterations of colour go by the general name *maculæ*. It is in the layer of young cuticle, called *rete mucosum*, that the colour lies which gives the peculiarity to the dark races of man, but some pigment is found in the papillary layer of the corium. If you examine our specimens of the skin of the negro, or make sections for yourselves, you will see immediately beneath the epidermis, and corresponding to the papillæ of the skin, this dark layer of cells.

In various morbid conditions of the skin, a pigmentary deposit takes place in the same position; but the most remarkable is that known as *melasma supra-renale*, or Addison's disease, where sometimes the whole integument becomes coloured, so as to resemble exactly that which belongs naturally to dark races, closely resembling the colour of a creole or quadroon; indeed, in some cases no difference could be observed between persons so affected and those born with an admixture of dark blood. You may therefore, if you should have any doubt upon the matter, clearly distinguish such a case from one of mere superficial staining of the skin by making a section, and observing whether the dark layer is under the superficial epidermis, seated close down upon the true cutis, and running everywhere in the course of the papillæ. In this microscopic section you will see the peculiar colour, or in these drawings taken from it. Although the name melasma has been given by some, the term *bronzing* or, in Germany, *bronze haut*, has been adopted by others, as the colour is of a greenish-brown hue. The colour is universally diffused, and is not in patches; but the bends of the joints, the scrotum, the areolæ of the breasts, &c., grow much darker than other parts. The colour varies a little from day to day, apparently through variations in the vascular supply. Mr F. Churchill, through the use of ether spray, produced temporary, and once permanent, blanching of the spot it acted upon, but we could not do so in a well-marked case; the dark colour persists after death. This colour is often mistaken for jaundice, though it is very unlike the common yellow hue of jaundice. But in very long-standing obstructive jaundice you see the skin wear a very dusky hue; it is, indeed, pigmented with black pigment in much the same way as in Addison's disease. This blackish change in jaundice deserves more attention than it has yet received.

In pregnancy a similar darkening occurs in some localities, as the face, areolæ and linea alba. It has been met with, also, once or twice in disease of the pancreas. This blackening is called Nigrities, so is a similar pigmented state found on the scrotum. The name *chloasma* is given to patches of a size from that of a crown piece to the palm of the hand, or larger, on the face or trunk, or especially on the extremities; these have been found connected with chronic hepatic disease, syphilis, and other disorders.

The name *Ephelis* is applied especially to a temporary discoloration of the skin from exposure to the sun, as the name implies; while *lentigo* is used for permanent *freckles*. The term *pityriasis* has been used rather loosely to denote a discoloration of the superficial epithelium, to which various trivial names were added, according to the colour of the blotches. Pityriasis *versicolor*, the variety which is distinctly recognised, is due to a vegetable growth (p. 269).

There is another remarkable staining of the skin, which is called *Ephelis ignealis*, or *ab igne*, and which you see represented in these models. It appears to occur in invalids who have sat for a long time before the fire and scorched their legs and arms; especially the former. You will see a number of reddish or brown lines coursing along the front of the leg, in the position of the superficial veins: the fact of lines exactly resembling these occurring as a post-mortem appearance, due to an imbibition of the colouring matter of the blood in the surrounding texture, would make it appear as if some similar cause was in operation in these invalided persons—that, in fact, some extravasation occurred from the vessels through the heat, so that a permanent discoloration took place in their course.

Discoloration arising from external use of *nitrate of silver* is very remarkable, and when once seen can never be forgotten, as the black-lead-like half-metallic lustre of the skin, and especially the face, is very striking. In these cases the internal organs have also been found discoloured, *e.g.* the mucous and serous surface of the alimentary canal and parenchymatous organs.

Albinismus is the state where colour is congenitally altogether wanting in the skin, as well as in the hair, eyes, &c. *Vitiligo* is the term formerly employed for an acquired and partial want of colour, but the word *Leuke* is now more generally used. Such conditions sometimes are so extensively diffused as to make the individual look "piebald." It is often associated with a deepening of the colour in the intervals, making it difficult to say whether the whole appearance is due to local excess or deficiency of the pigment; other conditions we shall mention when we speak of leprosy.

Parasitic Growths.—*Pityriasis versicolor*, or *chloasma*, is due to a parasitic fungus, known as the *microsporon furfurans*, discovered by Eichstedt. The disease is a very common one, and easily recognised in those buff-coloured patches so frequently seen covering the chests of the poorer people, especially of those who constantly wear flannel next the skin, and do not indulge in habits of cleanliness. If some of these yellow parts of the skin be scraped on to a glass slide, and the latter be placed beneath a microscope, the epithelial scales will be seen covered with the sporules of the fungus. In order to discover them you should place on the specimen a drop of water, and then a little liquor potassæ, or carbonate of soda, in order to render the scales transparent, when the sporules are readily seen. These are small round globules adherent to the scales, and beside them you will see some long cells placed end to end, which form filaments, or the *mycelium*, as it is called.

CORIUM, OR TRUE SKIN

The anatomical condition of the skin is generally little considered in distinguishing and treating its diseases; and this, no doubt, is very natural when you remember that the cutaneous affection is often only a part, and sometimes a small part, of a severe constitutional disease, as, for example in the exanthemata. There are many affections of the skin, however, which appear to be strictly local, though, as to many other so-called skin diseases, it is difficult to say whether the skin alone is at fault, or whether the eruption is not rather a mere manifestation of some inward and more hidden malady. Whatever may be the cause, however, of a skin affection, it would be our place to show the anatomy of the disease, apart from any connection it might have with an internal disorder. Having said this, you might expect that we should demonstrate to you what the peculiarities are, but this, unfortunately, we are unable at present to do in all cases, although we have no doubt the real change in the skin, in all cutaneous affections, will at some future time be discovered. We can see how, in the exanthemata, a mere difference in the state of congestion (due, of course, to some anterior condition) may cause a variety in the character of the eruption—as, for example, in *scarlatina* and *measles*. In the former, as we have before said, the congestion implicates the minute capillaries of the surface; in the latter it affects deeper-seated veins, whose fields appear as distinct dull blotches; and in this way, no doubt, a certain fixed anatomical condition exists in scarlatina, another in measles, another in *roseola*, &c. In *erythema* the redness is still more even. In all these rashes the blood is still held within the vessels of the skin; but if the blood be diseased, and the due relation be lost between the vessels and the tissues, then it bursts forth, and produces small effusions in the superficial layers of the cutis, known as *petechiæ*. Scarlatina, measles, smallpox in its first stage, may assume this character, but it is in typhus fever that it is more commonly seen. In this disease, the dark-coloured spots of the *mulberry rash* (as it is called), after a few days, cease to fade on pressure, and towards its termination, especially if it be fatal, the blood actually transudes from these congested spots, and petechiæ are produced. In the rash of enteric fever, the rose spots never go beyond the state of congestion; the blood being still in them, so that it is driven out of them by pressure. If blood transudes without any known cause, the name *purpura* is given to the disease. Writers on skin diseases class all these conditions, with discolorations of the skin, as "maculæ."

We have considered the rashes as inflammatory irritations with only the anatomical results of active fluxion in their simpler and milder forms. But sometimes exudations do take place, and then we can call them none other than inflammatory. Thus, in scarlatina, you have often observed raised papules on the skin, and occasionally in these a small quantity of fluid is formed; and probably, even in all such acute papular eruptions, even if no fluid is seen by the naked eye, some exudation really is present.

In fluxion to the skin, however, which constitutes a rash, there is usually simply a hyperæmic condition of the vessels; but in another class of cases, where the skin itself appears to be the seat of primary irritation, often from external causes, you find a more severe action in the corium, producing a papular eruption, being at least one form of what was called lichen. Dr McCall Anderson would, at the present time, bring this papular irritation under the description of eczema as inflammation of the corium, still calling it *lichen;* but we think Hebra's plan of limiting the term lichen to the more chronic papular epidermic heaps we described (p. 266) is far preferable. This form of apparently solid little elevations in acute cutaneous irritation will then take its proper place, as belonging to the early stage of *eczema*.

The next stage of eczema beyond this is shown in an actual production of serum. You may have watched such a process on yourselves; for example, when the hand is exposed on a hot summer's day to the sun's rays, you will first perceive a redness on its surface, and this is accompanied by heat and pain; you may afterwards perceive a lichen-like papular eruption (in the East called *lichen tropicus*, or prickly heat), at a later stage, small vesicles of eczema "piebald." It is often associated with a deepening of the colour in the intervals, making it difficult to say whether the whole appearance is due to local excess or deficiency of the pigment; other conditions we shall mention when we speak of leprosy.

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When the *true skin* and subcutaneous tissue are inflamed throughout, *phlegmonous erysipelas* is produced. In *erythema*, or superficial erysipelas, before mentioned, there is merely a swelling and slight exudation in the form of vesicles or bladders beneath the cuticle; but in the phlegmonous form, the whole depth of the skin and subcutaneous cellular tissue may become involved, so that purulent matter is formed in the deep parts, and much of the texture may die and require to be discharged in the form of a slough. A local form of a like inflammation is seen in *furuncles* and *anthrax*. You must not think these always commence in the deeper parts, for you will often observe that they begin on the surface and spread inwards; the sufferer first perceives a redness and feels an itching, and then presently a small vesicle forms, the whole being superficial; but in the course of a few hours, the swelling and induration has increased, until he discovers that the skin is considerably involved, when suppuration occurs, perhaps, with the discharge of a slough.

The distinction between furunculus and anthrax, boil and carbuncle, is usually drawn by the number of openings formed, single in a boil, multiple in a carbuncle. Hebra, we think, improves on this distinction by making the presence of a slough the sign of an anthrax or carbuncle, while the boil or furunculus only suppurates without sloughing. Either means of distinction is artificial, for the two conditions graduate into each other indefinitely. A large carbuncle brings with it danger of exhaustion or pyæmia. Boils are generally rather troublesome by their repetition. This may be due to some external irritation as when they arise in and complicate eczema, scabies, prurigo, phthiriasis, &c., or else it may be founded in a special disorder, of which it is the sign, when it is called *furunculosis*.

We must draw your attention to the terrible form of boil on the face generally seated on the lips, sometimes called *malignant carbuncle*, the facial veins become implicated in this disease, and suppuration extends along these to the cranial sinuses, causing death with cerebral symptoms sometimes rather suddenly.

Here, too, we must mention those likewise formidable kinds of cutaneous inflammation which arise by the absorption of *animal poisons*. These are first the *malignant pustule* of the hand or other part of the upper extremity, which forms as a bleb with a hard and extending base, from which the lymphatics inflame and may suppurate. Next the *necrogenic dermatitis*, which appears either from a puncture, or often without one, as a pustule, about the root of a hair, which may be small or as large as a little bleb. The base of this hardens, and it likewise implicates the lymphatics and perhaps the glands. Besides this more acute form, repeated similar irritations of lower degree will produce an obstinate wart-like swelling, of which you have some models here. This have we called *verruca necrogenica*. The third is *glanders*, a disease produced by inoculation or contagion from horses affected with it (p. 263). The skin here shows brawny patches, on which blebs and boils form, as in these models. The glands may swell, a condition which marks the variety of glanders which is called *farcy* but is essentially the same disorder. In one case we have found the muscles of the extremities full of small abscesses. A swift and grave form of pyæmic pneumonia is generally the cause of death. It is uniformly fatal.

Morbid Growths.—Several of the diseases which we have placed under this head might, we think, with almost equal propriety, have been regarded as chronic inflammations; but the same difficulty is found in the skin, as elsewhere, in defining what is an inflammatory product and what is to be considered as a tumour. For instance, we have just now spoken of phlegmonous inflammation of the skin and cellular tissue. But several chronic affections which we are about to consider might be looked upon as a slower process of a somewhat similar kind; indeed, the results of inflammation are so much like tumours that they often cannot be distinguished.

Here, for instance, is a drawing of an arm, showing a cicatrix following phlegmonous erysipelas, but from the omission of an explanation of it it had been considered as a case of keloid, a disease which is eminently chronic, but which, in the course of time, produces the same wasting of the cutaneous tissues and cicatriform appearance. In this form of disease, which we used to call here the *keloid of Addison*, and which is represented in several models and drawings, there appears to be a chronic formation of lymph or inflammatory matter in the skin and subcutaneous tissue, so that a swollen or brawny appearance is produced, as you see in this arm. This condition is now called *scleriasis* by Dr Fagge; it was first described by Thirial under the name *sclérème*. It produces a rigid state of the part without fever or other signs of active inflammation, it generally spreads from the spot first attacked, and is more common on the thorax and upper extremities. In this first stage the chronic inflammatory nature of the affection is evident; but in other cases this first stage may not be seen, for an absorption or contraction quickly follows, and thus the disease passes into a condition exactly resembling cicatrix, so that the marks on the limbs present just the appearance of a healed burn; indeed, not only are cases of keloid often regarded by the inexperienced as cicatrices of burns, but, as we just now mentioned, a real cicatrix might be mistaken for keloid by the partially informed, an error we have, indeed, witnessed in several cases. Here is a model of a scar-like patch on the forehead. It is called *morphæa*. Dr Fagge gives reason to think it is a scar of scleriasis. Mr Wilson thinks such patches, which he calls *vitiligo*, may be due to the resolution of lupus without ulceration. We shall presently tell you that Virchow uses the name morphæa for a stage of leprosy. It was long ago noticed by Drs Alderson and Sedgwick that Addison's keloid may quite disappear, and a case is given in 'Path. Trans.,' vol. xv, in which the marks disappeared, leaving only a pigmented patch. Dr Fagge has recently given other cases proving the temporary nature of the affection, among them some of Addison's own cases. Hence the disposition is now to cease using the word keloid for the scar-like affection described by Addison, and limit its application to the *keloid described by Alibert*, which consists not so much in this cicatriform appearance as in the production of large circumscribed growths. These are peculiar; they project from the skin, are of a bright red colour, and shining; they are vascular, and when removed are found to consist of a dense fibre-structure springing from the skin. These tumours often send off claw-like processes into the skin; but as in some instances contraction ultimately takes place, an approach to the other variety is produced. Dr Hillier described a case of Alibert's keloid undergoing spontaneous cure.

There are one or two forms of disease of the skin presently to be mentioned which link this affection to true *leprosy* or *elephantiasis*. Unfortunately under this name two different maladies have been described which are pathologically quite unconnected with each other although both occur in hot climates, and very often in the same locality, as in the West Indies.

The one is a local affection, the other constitutional; in the one the part affected, usually the leg, swells to an enormous size during a period of several years, while the patient is in good health; this is the 'Barbadoes leg,' originally called *elephantiasis Arabum*, and called in our museum *elephas*. If you look at this leg you will see that its right to a title founded on likeness to an elephant's foot is beyond dispute. The disease is due to a simple increase of the skin and subcutaneous tissue, whereby the former is hypertrophied and an immense thickening is produced beneath it, and, at the same time, the fat is often much increased in quantity, although it is sometimes much diminished, being compressed by the new-formed fibrous tissue. The skin also becomes brown, as you see in our models, and a number of fissures occur, which subsequently enlarge to oozing ulcers. This disease of the leg, as we have said, attacks those living in hot climates, especially the lower orders of the people, and mainly those of uncleanly habits. It commences as an œdema, and is then curable with care. It is probably the same disease as affects the genital organs in India, producing elephantiasis scroti. Although the disposition to it becomes excessive in the tropics, we are by no means free from the disease in this country, both as to the legs and genital organs. During the last two years we have had three cases of this affection in the hospital; the disease was of many years' duration, and the general health of its subjects did not suffer. In two of these cases, after amputation (the specimens may be seen on the shelves, as well as another of the true West Indian variety), we had an opportunity of making a section and observing the structure; the cuticle was very much thickened and of a dark colour, the cutis was also thickened, and beneath there was a quantity of adventitious fibrous material and fat, but no elements different in kind from those normally present. The bone was healthy, and the veins leading to the diseased parts were exceedingly thickened. The state of the lymphatics was not made out.

The other disease, which was called *elephantiasis*, was distinguished as *elephantiasis Græcorum,—leprosy*. It occurs in the tropics; but it is not limited to hot climates, being a terrible scourge in some districts, chiefly maritime, in Norway; indeed, the fullest account we have of it is from the celebrated work of Daniellsen and Boeck, which embodies the result of an inquiry ordered by the Swedish Government. To avoid confusion it is much better to confine the term elephantiasis to the local affection just described, and to name the disease under present consideration by the term *lepra*. This term is, indeed, now very generally so used, and its old employment for a variety of psoriasis is done away. The prevailing opinion is that this lepra is allied to, if it be not the same disease, as that with which the ancient Jews were afflicted, though Hebra gives curious reasons for the opinion that this was often scabies. True leprosy, as met with in the East and West Indies and in Norway, &c., appears under two forms, though these are not so separate but that they are found on the same individual. In the first form protuberances appear on the face and elsewhere. This kind is popularly called leprosy, or in scientific nomenclature *lepra tuberculosa;* the other form is attended not so much with swelling as with stiffness of the limbs and subsequent wasting and falling away of the digits; it is known familiarly as the *joint evil*, but, owing to the numbness which attends the disease, it is scientifically called *lepra anæsthetica*.

The first form, or tubercular leprosy, is occasionally seen in the hospital. The face and hands are chiefly affected. The anatomy of the disease shows the presence of a large quantity of a new formation in the tissues of the skin, extending from the surface of the corium down into the subcutaneous texture. The new matter is composed of cells of round or spindle shape, which, when seen in crowds, look like pus-cells, but in the spreading margin are larger and caudate. You see them well in this drawing from a case of leprosy in Stephen Ward two years ago. Virchow placed this leprous tissue in his class of granuloma or tissues resembling granulation-tissue, along with syphilitic tubercles and lupus, which superficially so much resemble leprosy. The new matter forms knots or tubercles in the skin, the cuticle over these knots becomes shiny and thin, and the glands and hair perish. But the disease rarely invades the scalp; it is only the brows and cheeks that become bare. When thus affected the skin becomes swollen and indurated, while protuberances form on it, so that the most frightful and loathsome appearance is produced; the face expanding and the features becoming bloated, with a grim and sinister aspect, better deserving the name *leontiasis*, which is now given to it, than the old term elephantiasis; the lips become much enlarged, the alæ of the nose expand, the eyelids thicken, and large folds of skin hang down on the cheeks and forehead; the hands and feet are generally similarly affected. The disease is not, unfortunately, altogether unknown as arising in our own country; but some believe that all such cases are only forms of syphilis. A few years ago, however, a very interesting case of true leprosy occurred in a man who had never been out of London; the models and drawings of his face you here see. It was affected in the manner we describe, producing this frightful disfigurement. He died with an ulceration of the larynx, a complication which is generally the immediate cause of death in Eastern climates. Very often, the skin of the whole body, although not thickened, is discoloured, and covered by white and brown spots. Virchow adopts the old word *morphœa*, *alba* and *nigra*, for these blotches, and considers them as due to the resolution of milder attacks of the leprous disease. Daniellssen and Carter have shown that the skin is thickened with new matter at these spots. The word *morphœa*, as we just now remarked, is used in our museum for circumscribed scar-like patches, generally on the forehead, which patches, Dr Fagge thinks, are connected with scleriasis rather than leprosy. You must make a note of this ambiguity.

The other form, or *lepra anæsthetica*, is characterised by the shrivelling away of the fingers and toes, as you see in these models. In the first instance, a mere numbness or loss of feeling indicates the onset of the disease. This condition is shown to be caused by a cellular material formed in the trunks of the nerves. We have already mentioned to you the observations of Virchow and Carter on this subject (p. 256). The skin of the limbs, especially of their ends, is in this variety occupied by the morbid product more uniformly; it contracts upon the textures beneath and ulcerates, so that the bones are reached and phalanx after phalanx dies and drops off, and thus the fingers and toes shrink away, leaving nothing but stumps, as you here see, the general health of the sufferers, meantime, remaining tolerably good which mutilation may reach to the loss of the forearms. Some doubt has been, however, expressed as to the authenticity of cases where the head is said to have come off through a slight knock. For further particulars of this interesting disease we must refer you to the work of Danielssen and Boeck.

We cannot dismiss this subject without drawing your attention to some other obscure allied affections, which appear either as occasional wonders in our own or other countries, or which are found in limited localities under special names. Virchow's great work on 'Tumours' contains a most interesting review of these disorders, which he groups together as leproid, lupoid, or syphiloid, in his class of tumours having the structure of *granulation-tissue*. The principal of these is *Radesyge*, occurring in Norway; an affection generally of the extremities, in the integuments of which soft tubercles form and ulcerate quickly, differing from leprosy in being limited in area, larger, and many deeper seated, also in ulcerating more swiftly. Hebra can see nothing in these, but sometimes syphilis, and sometimes serpiginous lupus; and Virchow has, we believe, come now to the same conclusion, *Yaws* or *Frambœsia* appear as growths resembling condylomata in structure and position, being red raspberry-like warts, which ulcerate and lose their skin; they may group together into larger masses, called "mama yaws;" they are contagious, and heal sometimes spontaneously. The disease belongs to the tropics in the Old and New World. There is the same question as to their syphilitic or leprous nature, as in the case of Radesyge. Sydenham set them down as syphilitic, and there are many who would do so now. The *Sibbins* or *Sevins* of some districts in Scotland is more generally regarded as syphilitic; it is very like both yaws and radesyge.

Besides these better known diseases, there are other cutaneous tuberous affections which are regarded commonly as peculiar local manifestations of syphilis—confidently by some, while others think this doubtful, because often no history of syphilis exists, and when it does there are perhaps no other cutaneous results of the syphilis, and there is a long interval since the primary disease. Yet further, there are on record peculiar forms of tubercular affection of the skin which you may find it useful to have borne in mind when some very surprising case occurs to you. Thus, there was recently a man under Mr Durham's care in Lazarus Ward, on whose skin were very remarkable tubercles and tubers, some of large size. They were red and shining, and rose rather boldly in relief; when larger, their surfaces ulcerated, producing altogether a very peculiar appearance of large spongy masses, with a soft-looking, grey, ulcerous surface. Some of them healed spontaneously, others dipped deeply into the subjacent textures, causing frightful gaping sores. In the end the poor fellow died worn out, and we found the tissues of the masses to correspond exactly with the "granulation-"

structure of lupus and lepra. This case is evidently of the same nature as those allied to frambœsia, described by Alibert under the name of *mycosis fungoides*. Anti-syphilitic treatment had no important effect on the disease. Very similar must have been the appearance of the Irish button scurvy, which, however, was contagious, or of the Aleppo evil, Delhi boil, &c., which are ulcerating cutaneous tubers, thought to be due to drinking unwholesome water; but we have never seen these.

Fibrous deposits appear in the skin and subcutaneous tissues in the form of *syphilitic tubercles*, which may sometimes, as we have seen, resemble leprosy very closely. These have the characteristics of gumma in other parts. You must note that they are tubercles, not in the same sense as that in which we speak of tubercle in phthisis; but in the sense in which the dermatologists define tubercles, merely from their external form; thus, "solid swelling of the skin; size from a lentil to a hazel-nut, and covered with epidermis." You find such tubercles also very often in *acne indurata*. Fibroid material makes up more or less of them as a new matter uniting together the inflamed and suppurating sebaceous glands.

Many different looking forms of disease are included under the name *lupus*, so that it is difficult to define the term. Lupus is generally divided into *exedens* and *non-exedens*. The exedent form is not clearly distinguished from the so-called "rodent ulcer," and in this a cancerous growth has been shown by very competent observers to exist just under the spreading edge of the ulcer. In lupus exedens of the face a cellular growth of granulation-tissue is found beneath the surface. The disease usually spreads slowly, but may become phagedænic or gangrenous, first destroying the soft parts, and subsequently the bones. In the non-exedent form, which we are every day meeting with, there are also many varieties; in all these there is a morbid formation, which in one case is followed by a slight superficial ulceration, as in the *scrofulous* form; in another by a desquamation, as in the condition called "*psoriasis lupus*"—lupus exfoliativus; another variety has been called *lupus erythematodes;* in this the surface is evenly swollen, with a smaller quantity of the granulation-cells. In lupus non-exedens a quantity of newly formed, round, and spindle cells, like those of granulations, fill up the areolæ of the connective tissue in the corium, destroying the sebaceous glands and the hairs, and causing the cuticle to become dry and scaly; or else throwing it off, and breaking down into an ill-conditioned ulcer. Time will not allow us to enter more fully into the details of these and other cutaneous diseases, and we therefore must satisfy ourselves with this outline of their general character.

In *molluscum fibrosum* pendulous tumours grow from the skin, sometimes in great numbers. They are described by Virchow as composed of tegumentary tissues and allied to elephantiasis. But in a case we recently inspected Dr Fagge found a ramified sebaceous gland in each small tumour. Such a large gland is held to characterise *molluscum contagiosum*, and to be absent in the fibrous form, yet the large tumours in this case were fibrous. The subject needs further investigation. We have already spoken of a state of the thigh resembling elephantiasis in which the lymphatics are likewise enormously enlarged, and may burst, producing lymphorrhœa.

Condylomata are new growths of the papillary layer of the corium; they, too, consist of tissue resembling that of granulations. They will be described in the surgical lectures. Two kinds are distinguished; the first, called broad condyloma, or mucous tubercles, are the wide, flat, and softer varieties of papillose elevations of the corium; these are generally met with near the genital organs in connection with syphilis. The other kind are the acuminated condylomata, which are not specific, but due to irritation from discharges; these are of more slender figure, and made chiefly of epithelium. Among them *warts* are new papillary growths of acuminated form, consisting of masses of epithelial cells growing in the form of a cone, in the midst of which is a loop of blood-vessel. Sometimes, as you see in this drawing, one cone sprouts out of another, and this indicates their mode of formation; for, although the papillæ of the skin may, in the first place, determine the papillary form, yet afterwards processes shoot out and grow from the old ones—a fact which can be easily made out by careful examination. Although the papillary part is composed of epithelium, yet there is often a substratum of new fibrous structure, and sometimes an actual tumour, as seen in the warty growths from the labia pudendi.

We may here mention to you that curious condition called *vitiligoidea* by its discoverers Addison and Gull, but which is now known by the name of *xanthelasma*. This has recently been shown by several cases in the hospital to be a result of long-standing jaundice from any cause. Severe itching of the skin has usually preceded it. In some cases of jaundice we have known the whole epidermis curiously dry, so that a scratch made a chalk-like mark upon it. The xanthelasma appears in two forms, *x. plana* being an opaque yellowish-white patch, not elevated. The pigment in the patch is gone, and a quantity of fat-grains are present. In chronic jaundice this state affects the palmar surfaces of the hands, the eyelids, ears, scrotum, &c.; but you may meet with patches near the inner canthus on the upper eyelids, when there is no evident hepatic derangement. Mr Hutchinson has collected many such cases; they were known to and figured by Rayer. The other form is called *x. tuberosa*, from the presence of tubercle-like knots on the knuckles, elbows, &c.; these knots are composed of fibrous tissue, with a great quantity of granular fatty matter. Xanthelasma is not limited to the skin; we have found it in the lips, the gums, and the trachea; also similar opaque patches we have seen on the spleen, and in the lining of the gall-ducts in these cases.

Cancer implicates the skin both primarily and secondarily. When a cancer grows in a superficial organ, as the mamma or testis, or a lymphatic gland or bone, the skin becomes in time adherent to and infiltrated with the growth, looking coarse, dark coloured, and vascular, and feeling hard. This is often one of the most important signs of the malignant nature of a growth.

The commonest examples are met with in extension of carcinoma mammæ, especially after the removal of the breast, when the disease may be seen returning in the skin in and around the cicatrix, as a number of hard nodules, which, when examined, will be found to show compact epithelioid cells imbedded in the meshes of a fibrous matrix, which is principally at least composed of the natural elements of the skin itself, so that when the growth has sprouted out further away from the integument, it is softer, the matrix then being an altogether new formation.

But the proper cancer of the skin is *epithelial cancer*. The appearance of this is peculiar when seen with the naked eye; instead of showing a vascular juicy structure, like ordinary cancer, it is dry, friable, and has a curdy look. The main seat of all such growths is an epithelial surface. In the skin it generally affects the papillary layer, and then the papillæ grow out into warty forms often very complex. The warts may be dendritic and widely spreading, or they may unite to form a horn-like sprout not to be mistaken for the simple epithelial horns we have before mentioned. In other cases, but more rarely, the cancer is in the deeper layer of the cutis, and then the papillary layer spreads with no warts or but few warts over the globose or conical mass of the cancer. When you examine any of these epithelial cancers you find, in sections of the warty layers that the slice is composed of various conic sectional figures from the cut papillæ, with some old shed epithelium and dirt filling the occasional intervals where the papillæ were not in close contact. The papillæ are composed of a central vascular core, around which are large flattened epithelial cells with large polynucleolated nuclei, and with edges toothed to fit each other. The central core is also, except the vessel, composed of epithelioid cells, but not arranged concentrically, and sometimes we have found the core of granular elements, *i. e.* lymphoid cells. The part beneath the papillæ shows epithelioid cells of more irregular shape and size, and forming together much more irregular figures than the sections of the papillæ; still there is always in epithelial cancer a structural configuration of the elements into tubes and knots very characteristic. There may be abortive hairs or gland-tubes or lymphatic channels full of cancer-cells (see Plate V);

it is here in the subpapillary part of an epithelial cancer that you find the laminated corpuscles which are so characteristic of this kind of cancer. These appear as gatherings of epithelial cells in layers tightly packed around a circular or oval central space, the whole very like the section of an onion or like a bird's nest.

We also meet occasionally, though very rarely, with diffused primary scirrhous cancer of the skin; this takes at first the form of a wide dusky, purple, brawny patch. It was thus in the case of a woman in the hospital, whose skin and subcutaneous tissue were indurated and brawny, and in some parts elevated into a number of small tumours, presenting the appearance seen in this model. The whole trunk being affected, it could scarcely be said with certainty how far the new tissue was fibroid and allied to keloid or scleriasis, or how far such a slow growth was carcinomatous, although, after some time, it was tolerably clear that it was of the latter kind. The woman dying some months after she left the hospital, a post-mortem examination revealed cancer, not only in the skin, but in the bones and other parts. Such is a good example of *scirrhous cancer*. Another equally good one furnished this model, where you see a large dusky thickening on the front of a man's chest, which rapidly opened and proved to be scirrhous cancer. When a circumscribed prominent growth proceeds from the skin some distance as an isolated tumour, it generally is structurally a *medullary sarcoma;* if very vascular, it is what was formerly called *fungus hæmatodes*. We must again make the remark, which we have repeatedly made before, as to the difficulty of defining many forms of maladies, seeing that they pass by insensible degrees into one another, and thus you may conceive, that if a cancerous growth were very slow in its formation, and were of the fibrous kind, how much it would resemble keloid, and some other diseases already mentioned.

Melanotic cancer of the skin is a pigmented form of sarcoma generally composed of spindle cells with more or less of an intercellular substance, that may or may not be fibrillated, but generally is so. The pigment is in the cells; sometimes it is very unequally distributed in them. It is very remarkable that often in these cases the pigmented character of the disease is determined by the previous presence of pigment in a mole. Thus, in this drawing you see a black tumour on the arm which originated in a mole, and the disease after excision soon returned in the axillary glands, as cancer combined with pigmentary matter. In some cases, however, the disease has no such evident origin; you see here a piece of skin containing melanotic tubercles; this is the drawing of a man who was similarly covered, and who had the disease also in his internal organs.

Nævus we have already spoken of, and allied to it are mothers' marks and dark hairy patches, as you see in these preparations.

In connection with the integuments you will meet also with fatty tumours, or *lipoma*, of which you will see several large specimens on the shelves. Also various forms of fibroma and sarcoma; the former composed of simple fibrous tissue, which appear to be altogether innocent; the latter composed of nucleated fibre, which often *recur* after removal. These have been called *fibro-plastic* or recurrent fibroid (Plate II). You likewise meet with myxoma, a tumour of a softer consistence and gelatinous appearance (Plate III). These all differ from cancer, in being circumscribed by and not involving the neighbouring tissues. We have also met twice with pure *adenoma* (Plate IV) of the skin and subcutaneous tissue, the structure being identical with that of the breast in lactation. One of these was partially charged with lime salts.

We might here mention gouty concretions of urate of soda met with in the teguments under the name of tophi, chiefly in the ear, in the scrotum, or about the elbows, &c. The same deposit also invades the teguments from gouty joints.

Parasites.—Of animals that inhabit the skin the most common in our country is the so-called itch insect, the *sarcoptes*, or *acarus scabiei*. This is not a true insect; it belongs to the class arachnida; it has a rounded body, with eight legs, the four anterior being armed with suckers, and the four posterior with bristles; it has two pairs of mandibles armed with teeth. The female is larger than the male. The female itch mites burrow in the rete mucosum, and lay their eggs as they work their way along, so that at the end of the burrow is the animal herself, while the eggs, in their order of juniority, lie in the tunnel behind her. Besides these tunnels or 'cuniculi' there are usually eczematous vesicles due to irritation and the effects of scratching on the skin around.

Another creature, of an allied species, is the *harvest bug*, which some suppose to be an *ixodes*. Many of you may have been attacked by these tormenting creatures while walking through stubble fields in the autumn; by scraping the place which itches, the insect may be caught and put on a piece of white paper, when it appears as a minute red body.

In the West Indies a most tormenting creature is the *chigoe*, or *pulex penetrans*. The female penetrates the skin, and there lays its eggs, producing in consequence an irritable sore. It is very like our common flea, but smaller.

The *Dracunculus medinensis*, or Guinea worm, is occasionally seen in the hospital, in sailors who have come from the coast of Africa and some other tropical climates. It is a long, thin, viviparous worm, belonging to the nematoda. It measures several inches in length, as you may see in any of these specimens which were removed from the tegument of the body; the end protruding from the skin was secured, and the remainder of the worm gradually coiled out by slowly rolling round a piece of paper. It is supposed that they enter when very small through the ducts of the skin, taking advantage of opportunities when people are bathing, wading, &c., or they are found in the legs of those who have been occupied in boats where water has lain at the bottom, or in the backs of those who have been employed in carrying water; they come in the rainy season, and attain their full size in about a year.

We must here too mention to you the curious "fungus disease" of the foot, Mycetoma Carteri, for the first explanation of which we are indebted to Dr Vandyke Carter, of Bombay, before whose observations the disease was supposed to be scrofulous, though its superficial characters had been described. In this affection the foot is found swollen, and having crater-like orifices on its surface, which lead into channels of about the size of a quill. These permeate the textures of the foot, traversing its tissues, bones as well as soft parts. In these channels is a fish-roe or poppy-seed-like substance, which may be of a light greyish colour, as in the variety called "madura foot," or may be brown or black. In the latter case the microscope shows the presence of a fungus:—Chionyphe Carteri. There is no such fungus in the pale-coloured variety. In either case the substance has the form of clusters of little balls of an amorphous substance, each ball surrounded by a covering of radially placed, flexuous, cilium-like filaments, which Dr Carter says are fatty crystals, but this being so their constancy, evenness, and regularity are very curious. The absence of the fungus in some cases is as certain as its presence in others, and the difference is not yet, we think, fully explained, but Dr Carter considers the colourless form to be the remains of the fungus in an altered condition.

SUDORIPAROUS GLANDS AND DUCTS

We have not much to say in reference to these organs, but we may mention in connection with them the morbid condition known as *miliaria* and *sudamina*. Hebra distinguishes between these: sudamina, he says, are only swellings of the hair-sacs and mouths of the sebaceous follicles in cases of excessive heat and sweating. The name miliaria, on the other hand, he reserves for the same condition for which we employ it. Thus, in febrile affections a clear fluid collects beneath the cuticle in small transparent drops; this fluid is often acid when tested, and so it differs from the inflammatory exudation in most vesicular diseases, as eczema, &c., where the fluid is alkaline. Hebra denies the acidity of miliary eruptions; nevertheless in acute rheumatism especially it certainly is usually acid.

SEBACEOUS FOLLICLES

The abnormal state of the sebaceous secretion gives rise to several important diseases of the skin. Thus, the secretion, being dry, plugs the mouth of the follicle, and then the follicle becomes distended with a mixture of secretion and epithelial scales drier than the natural secretion. This constitutes *comedo*, with which you are familiar as a little elevation with a black top, very common on young gentlemen's faces during the active sprouting of the beard. Where the skin is very thin, as in the eyelid and scrotum, the collection occurs in the form of a little white knot with the layer of skin running over it; this is called *milium*.

If an acute inflammatory product is formed around a comedo you then get *acne punctata*; or if the inflammation is severe around many follicles, especially in the dense tissue of the nose, *acne rosacea*. If, instead of acute inflammatory formation, a slow fibrous growth surrounds the follicle, swelling in the direction of least resistance, so as to rise off the surface and acquire a neck and at last a pedicle, then you have the pendulous sebaceous tumour of *molluscum contagiosum*, the contagious nature of which is, however, much disputed. Hebra denies it. Virchow admits it in a modified way, saying that where there is foul secretion from the sebaceous glands of one this is likely to irritate the sebaceous glands of another much in close contact, as a nurse child, for instance. Here is a model showing the usual example of such tumours around the mouth of a child. Mr Hutchinson has published a similar example.

Sometimes the process by which comedines are formed reaches a new scale of magnitude, so that the cyst grows very large, as large as a walnut, an apple, a cocoa-nut. Such tumours are called *wens* or *sebaceous tumours*. Sometimes, as by Boerhaave and Sir Astley Cooper, the opening of the follicle is discovered, and the origin of it thus declared. Sometimes a fine cord has been found connecting the cyst with the skin and representing the obstructed duct. In by far the greater number of cases, however, the skin over the tumour is tense and shining, and no opening can be detected, so that these cysts are looked upon as new formations by some. Such cysts have a very thin wall of fibrous tissue from the corium; inside this is a thick compact layer of epidermic scales, with more or less fat between them. The youngest layers next to the cyst-wall are best formed. The older are more broken down, and form a pultaceous mass in the centre, from the consistence of which Virchow adopts the old name atheroma for these tumours. Sometimes the epithelial cells lie in layers, and between them are crystals of cholesterine, the whole forming a pearly mass called *cholesteatoma*, very similar to the tumour we mentioned as occurring sometimes in the membranes of the brain. Fine pale hairs are sometimes found in such cysts, as in similar cysts in other parts, mingled in the fat, and when these cysts are small, and mostly in young subjects, you may find the lining of the cyst to be much like skin and to have hair-bulbs in it. Similar cysts bearing hair are found in the skin of some of the lower animals, the hair being like that natural to the particular creatures. Mr Cock drew attention to the sprouting of growths from these large sacs after they have been opened, and a formation of warty growths in the interior of smaller epidermic cysts has been noticed by several observers. Sir J. Paget points out the hereditary descent of these cysts in families. In thinking of them with a view to operation you must remember the dangerous inflammation which surgical interference with them is apt to excite, a fact attested still by the general experience of the profession.

NAILS

Ungues adunci, or curved nails, in connection with phthisis, were observed as long ago as the time of Hippocrates. This condition is found on the toes as well as the fingers, and is not peculiar to consumption, as it is sometimes seen in heart disease, especially of the aortic valves, and sometimes in bronchitis. It reaches an extreme degree, too, in cases of empyæma; it appears to depend, partly at least, on venous obstruction, especially when associated with hectic, but its causation is not quite satisfactorily explained. The lowest degree of this change shows itself in a thickened shiny state of the fold of skin over the root of the nail. Next the nail is thick and curved. In the extreme degrees the whole end of the finger is obviously thickened; but anatomical examination has not shown anything more than slight general hypertrophy of the tissues.

Onychia and *paronychia* are very painful diseases, accompanied by suppuration, affecting the root of the nail, and often leading to its loss; they are described by the surgeon. In *onychia maligna* the nail is discoloured, loose, and recurved, and rests on an angry-looking sore, while the end of the finger is clubbed and dusky with congestion.

Some cutaneous diseases implicate the nails and their roots, as well as the skin. This is the case sometimes in eczema, impetigo, or in the less acute inflammation of simple psoriasis. Syphilitic psoriasis, acquired or congenital, also occasionally, though rarely, affects the nails, causing their surfaces to be ragged and irregular, while the skin around the nail is inflamed and red.

Mr Hutchinson has drawn attention, not only to the state of the nails in syphilitic psoriasis, but to a state of them in congenital syphilis, which is very interesting in connection with his observations on the teeth in this condition. The nails are symmetrically affected; they become dry and brittle, and so are fissured and broken at their free edges; the superficial layers are alone affected. This condition is well represented in Mr Hutchinson's plates in the Sydenham Society's 'Atlas.' There is, however, a more remarkable affection in the form of a chronic general onychitis, which you may see in the living subject, in the cases of two or three women now attending the out-patient room. The nails decay and fall off; they first become opaque and much thickened, and their substance is soft. The disease is due to an inflammation of the matrix, which is swollen, and readily bleeds.

Plate XVII of the Sydenham Society's 'Atlas' shows this condition very well, and in Mr Hutchinson's 'Catalogue of the Collection' he remarks on the very great symmetry of the disease, which shows its constitutional origin. He says it usually attacks children and young adults, and there is seldom any history of syphilis. In this our own observation agrees with his.

Dr Fagge has observed ringworm affecting the nails; the nail was ribbed longitudinally, and its colour was mottled with dark brown; the texture brittle. Microscopic examination showed *Tricophyton tonsurans* in considerable quantity in the substance of the nail.

The handling of subjects injected with arsenical solution induces an inflammation of the bed of the nail, which may produce suppuration under it. On one occasion we saw a long continuance of the action of arsenic produce a warty growth in the seat of such inflammation, which caused great pain for many months.

Attention has been drawn to states of the nails which sometimes appear after illnesses, and are of great interest, as revealing the general nature of a change during some obscure constitutional affection. These may be either bands of white colour across the nail, or bands of thickening of its texture, which, of course, move in the growth of the nail, so as to remain for months as evidence of what occurred.

The nails, if neglected, may grow very *long* and *curved* in all directions, as you see in the specimen.

HAIR

Colour.—Greyness of the hair depends on the presence of small vesicles of air in its shaft; the change begins, as is well known, on the temples, though in those prematurely grey we do not know that this is the case. Those who have investigated the subject give full credence to the stories related of the hair turning grey in a few hours from excessive grief.

Atrophy and *falling off* of the hair often occurs after febrile attacks, where, no doubt, the whole skin has been diseased. In various chronic maladies, as syphilis, &c., it partially falls off. Common baldness of elderly males has been plausibly ascribed to the wearing of tight hats compressing the vessels of the scalp. This natural baldness occurs, as you know, at the crown of the head, and extends equally from this part; but you sometimes meet with a disease in which the hair disappears in circumscribed patches—this is called *alopecia areata.* There may be only one patch or more; it may even spread so as to remove the eyebrows, whiskers, &c., in short, every hair from the body, producing *general alopecia.* This was formerly thought by Willan and Bateman to be dependent upon a pustular disease, which they call porrigo decalvans; but such a baldness is by no means always preceded by any affection of the kind, and the modern theory is, that the decay of the hair is due to a vegetable parasite, which has received the name of the *microsporon Andouini.* We think this is very doubtful; at least, we are among those observers who have never been able to discover a fungus in any of the hairs surrounding the bald patch, and therefore it has still to be satisfactorily shown what is the cause of the withering and falling off of the hairs in this manner. There is another affection of the hair, which we do not think is described in books, but about which you will be sometimes consulted, and that is a *breaking of the hair.* Owing to the fracture, a white spot is produced; and if a number of these are present, as, for example, in the whisker, they have been mistaken for ova of pediculi, and alarmed the patient. If you examine one of these hairs, you may find it broken in one or two places, and thus presenting a nodulated appearance; and if then examined by the microscope, the fibrous structure of the hair will be found to be broken up into a brush; we have seen interspersed a few dark granules, but we could never make out distinctly any sporules of fungi. You see examples of what we mean in these drawings. The *growth of the hair* varies much in health and disease. Curly stiff hair generally denotes health, while long straight hair breaking at its ends denotes a feeble and often phthisical subject. Such a distinction may be of value, with other signs, in assisting the medical man who has to examine a body as to the cause of death. If a patient has been long ill, and with a wasting disease, the hair is long and straight, both on the head and beard; but the condition is especially observable on the pubes, where the hair may be seen hanging in long straight tufts. In phthisis and some other wasting diseases there is often a disposition for the hair to grow, and thus the chest may have become recently covered with long straight hair.

Vegetable Parasites.—These have received a considerable share of attention, as it has been thought that some very important contagious diseases of the head are due to them. We have already mentione
one of these, the *microsporon furfurans,* as the cause of pityriasis ve
color. These fungi are all more or less alike in being co
simple cells and filaments; the cells are roundish or o

in shape and size in different species. If the cells are long and placed end to end, they constitute branching filaments, or a *mycelium*, as it is called; upon these is placed the receptacle, or *sporangium*, containing the reproductive *sporules*. The structure of these fungi is best brought out by the addition of a little alkali to the slide. The fungi are so much alike, indeed, that some observers, with Professor Hebra, believe they all have a common origin. This view is supported by the occurrence of favus in patches of tinea tonsurans. This association is shown in one of Hebra's plates, copied in the Sydenham Society's 'Atlas.' It has been suggested that the apparent tinea tonsurans was a variety of favus, but there is no evidence of this. Amongst the most important is the *achorion Schönleinii*, which is the cause of the favus, or, as you will still see it called in some of our drawings and models, *porrigo lupinosa*, the name used for it by Willan and Bateman. We refer to the disease characterised by large yellow, raised, circumscribed cup-shaped masses growing on the surface of the skin, and more particularly the head, the skin between being little affected or unaffected. If these crusts be examined, they will be found surrounding the hairs, and composed of large oval sporules and a mycelium, as we have just mentioned. They may be found in the follicle, but do not necessarily destroy the hair. The *tricophyton tonsurans* was first described by Gruby, in 1844, as the vegetable fungus which causes ringworm or tinea tonsurans, or, according to our old nomenclature, *porrigo scutulata*. We allude to that disease of the scalp where there are branny, desquamating patches covered with short broken hair. The sporules are found to have entered the sheath of the hair, and crept upwards into the stem, so that it decays and breaks off short. The sporules are rounder and smaller than in the preceding variety, and distinct filaments are not so easily seen. You will remark, that in this disease the parasitic growth gets into the fibrous structure of the hair itself, and not merely around it, as in favus. The *microsporon Andouini* we have just mentioned is a fungus believed to cause the falling off of the hair in alopecia, but this we have never yet seen. The *microsporon mentagraphytes* is another species which is found in the sheath of the hairs of the skin, constituting the disease known as sycosis. We have frequently pulled out hairs and seen the sporules, but we cannot say that they exist in all forms of the disease, and that the pustular eruption met with is always due to this parasite. Moreover, it requires a long practice with the microscope to be able to detect the differences in these fungi, which so much resemble one another; and we, for our part, do not know how this fungus differs from the trichophyton of ringworm already mentioned. It is stated also that a species of tricophyton may be found in the peculiar disease of the hair occurring in Poland, where it becomes matted together as you see here in this specimen of *plica*

Polonica. We are not at all decided that these parasites actually constitute the disease, and that they are not accidental; indeed, we think a vesicular eruption may often be seen to precede their appearance.

Animal Parasites.—These are *pediculi*, or lice. The most common are the *pediculus capitis* and *pubis*, the latter also called *phthirius*. The great difference between them is the length and shape, the one being long and the other of a squarer form. These are distinct species, and will not occupy the habitat of each other. Another species of phthirius affects the eyebrow. As lice are especially seen on sick people, it is thought a special genus attacks them, and this is called *pediculus tabescentium;* but of the truth of this we cannot speak. It is also said that the creatures which one sees crawling on the clothes of beggars form a distinct genus, the *P. vestimentorum*. The eggs of lice are attached by a gummy substance to the hair, where they may be seen sticking in rows when the hairs are plucked out.

THYMUS GLAND

Nothing is known of the function of this body, and almost as little of its pathology. It is said, sometimes, to be found *atrophied* in new-born children, showing that it must have undergone a change in fœtal life. Sometimes, in children, it is found large, and is said to be *hypertrophied;* in some cases we have seen the gland too large, but this has been due rather to a persistence of its earlier state, and not to any actual increase in size. It is thought, however, by some older German writers, as Hangsted, that such enlargement may cause pressure on the air-passages, and give rise to what they call *thymic asthma*, an opinion that would correspond with the well-known view of Dr Hugh Ley, who supposed that enlarged lympathic glands compressed the recurrent laryngeal nerves producing spasmodic croup. We have no experience of thymic asthma. Modern German writers, as Friedleben, have thrown much doubt upon it; Virchow holds it possible, and gives a case of fatal asthma in a child, wherein he found the thymus to weigh six and a half drachms. In later life the thymus should have nearly disappeared, as you know; but it has happened very curiously that a large thymus has been discovered in an adult with leukæmia, and also once or twice without leukæmia. Thus we met with a thymus gland weighing one ounce and a quarter in a man of thirty-two years, who died of purpura in Stephen Ward; his spleen was large as in leukæmia. It is interesting to see how pathology thus agrees with anatomy, in allying the thymus with the other blood glands. Some interest was created by Dubois's observations of suppuration of the thymus in visceral syphilis;

these at first received partial confirmation, but of late have fallen into some neglect, and we think doubt. We have seen the usual milky secretion in the thymus in syphilitic infants, but we have not seen pus. Abscess is a rare result of syphilis. Occasionally, tubercular disease has been found in the thymus, and, we believe, cancer. Among our preparations we have this one, of enlarged thymus, and this other, which is called cystic; but it is doubtful whether this is thymus at all.

THYROID GLAND

The most common form of affection of the body is its *enlargement*, as seen in *bronchocele* or goître, when it often reaches an immense size, but less degrees of its enlargement are constantly met with on the post-mortem table. Sometimes, these enlargements appear to be *simple hypertrophies*, the tissue being close and uniform; but very often the size is due to a jelly-like or colloid matter being formed in the interior of the organ, and sometimes the loculi dilate into large cysts filled with this gummy substance. The latter, when in great quantity, is fluid, but sometimes is as firm as wax. Several specimens are before you of these *hpertrophied* and *cystic* thyroid bodies. Besides this general change, very often distinct and isolated cysts are formed, growing from one lobe or the other, and attain a very great size; and sometimes the walls of these *ossify*, as you see in this specimen. Knowing that such are not uncommon, it is not surprising that an hydatid cyst which Mr Cock removed a short time ago from the thyroid should have been looked upon as part of a bronchocele. We have seen this body involved in inflammatory and suppurative processes in the neck, but not, we think, primarily so. Tubercle we have never met with, and *cancer* but rarely. It is generally agreed that cancer of the thyroid is very rare. Walshe estimates the proportion of thyroid to other cancers as 1 to 1000; we have found it only on three or four occasions, and in all but two the fact of the neighbouring lymphatic glands being implicated, made it questionable whether the disease had not commenced in them. This appears to be a specimen of the disease.

We must here consider the anatomical state of the thyroid in the curious condition known as exophthalmic goître, or Graves's disease, or more rightly Basedow's disease, since he first in 1840 recognised it as a special cachexia, and described it so fully that little has since been added. In this condition, when fully developed, the eyes are prominent, the heart irritable, and the thyroid large; we have already spoken of the healthy state of the eyeball. The thyroid equally shows no essential change; it is generally very vascular, and it varies in size. In thirteen cases collected by Virchow, six had the tissue normal, but hypertrophied, and the vessels large; in the other seven there were local changes, thus one had some gelatinous degeneration, three had cystic changes, another had hæmorrhage into the tissue in spots, and another a cartilaginous patch. Of three examples we have inspected, one had the gland normal in texture but large, the other two had slight local changes in the form of tumours of thyroid tissue. Thus, there is no known post-mortem condition of the thyroid gland proper to Basedow's disease. During life you will generally hear curious loud vascular murmurs in the gland and neighbouring part of the neck.

DISEASES OF THE RESPIRATORY ORGANS

AIR-PASSAGES

These are divided into the LARYNX, TRACHEA, and BRONCHIAL TUBES. In many cases we shall have to speak of these parts separately, but, as they are so often the subject of disease at the same time, we shall prefer taking the morbid conditions one by one; and then, if need be, show how each individual part is especially affected.

Dilatation.—A dilatation of the *trachea* has been mentioned by Rokitansky as occurring in its posterior part in old people, but we have hitherto failed to find it; and we therefore shall pass on to *dilatation of the bronchi*. This has been divided into various kinds, but we will only mention the *two* principal varieties, as they, for the most part, correspond to two very different pathological conditions. One is a *general* and uniform dilatation, and the other a *saccular;* the first is usually associated with a simply collapsed condition of the adjacent lung tissue, and is a primary affection; while the latter is connected with a structural change in the parenchyma, and is mostly a secondary process.

To commence with the saccular variety; we mean by this term a condition of tube where a tendency to sacculation is approached, and not where distinct sacs are formed, which is rarely the case. Such dilated tubes have long been observed in the upper lobes of the lung in connection with chronic pneumonia, and in the similar indurated condition of tissue in phthisis; in these cases, where a vomica has been suspected, but a mere dilatation of a tube found, it was formerly thought that the latter had preceded the induration of the tissue; but some years ago the opposite theory was propounded by Corrigan, that the disease of the lung substance preceded, and caused the expansion of the tubes. He studied the subject, especially in those cases where the lung was diseased throughout, and, being converted into fibrous tissue, was styled *cirrhosis;* but the same causation holds good where portions only of the lung are affected, as before said. This theory is, that owing to the chronic change in the lung, and its conversion into a new fibrous tissue, a contraction ensues, and that as a consequence the bronchial tubes are drawn open; just as during the same contractile process, the walls of the chest are drawn in. Thus, you see in these specimens of cirrhosis, the lung tissue is quite gone, and its place occupied by a dense fibrous structure, and through it are running these large irregular spaces which have originated in the expansion of bronchial tubes; the same thing you may see in most cases of phthisical lungs, if the disease be of any standing. In such cases you will constantly hear the question asked, whether the cavity or hollow space is one formed in the substance of the lung, or is a dilated tube; but it is very difficult to decide with certainty, and, indeed, it is often a mere question of words, for the cavity becomes so altered by dilatation, that the mere continuity with the tube is not sufficient to prove the point; for, supposing in the first instance a tube should dilate, it afterwards increases at the expense of the pulmonary structure, and in the mean time the original character of the bronchus is lost.

The other form or *general dilatation* of the tubes, although associated with an atrophied state of the pulmonary tissue has its origin in a bronchitis. It is found in long-standing cases of this disease, especially in children, where it has succeeded to a hooping-cough, as you see in this specimen, and which will explain the nature of the affection. This lung came from a boy who had suffered from severe bronchitis (or asthma, as it is popularly called), for many years, and the physical signs suggested cavities at the bases of the lungs; these organs, however, were in the condition now seen: the primary and secondary divisions of the bronchi are natural until the lower lobe of the lung is reached, when the tubes become expanded, and occupy the greater portion of its lower part like the fingers of a glove; they get wider and wider until they reach the bottom of the lung, when they abruptly end, being there as large as the primary branches of the bronchi themselves; other parts of the lobe show these large spaces or cavities, which are sections of the tubes.

In these cases the mucous membrane is generally red, swollen, and villous, and covered with secretion, whilst the walls are thin; in the other or saccular variety, the walls are thickened from being involved in the fibrous induration around them, the mucous membrane is thickened, and the longitudinal and circular fibres are hypertrophied; the circular passing as distinct ridges around the tubes.

You can see that neither form of dilatation can occur without an atrophy of the intervening tissue; but the relation which these two conditions bear to one another is still a subject of controversy. In the first-mentioned variety, where there has been a distinct history of chronic pneumonia or pleurisy, we must regard the change in the parenchyma as the starting point in the process, but in the second variety of general dilatation following a bronchitis, it is probable that the inflammation of the tubes has originated it. There are pathologists, however, who, following Laennec, maintain that under all circumstances the primary morbid state is in the tubes, that in one case a simple dilatation, with collapse of the parenchyma, results, whilst in the other a lobular catarrhal pneumonia following the bronchitis, and leading to solidification and contraction of the lung, brings about dilatation. That the first case is sufficient to produce dilatation is shown by a case which occurred here in the person of a lad who died of bronchitis nine days after an accident, and in whose lungs the tubes were found greatly distended; the parenchyma was healthy, and the condition found was no doubt a recoverable one. In cases of longer duration the intervening tissue may be found of a dark colour, tough, and airless; the air having been squeezed out, the lung has become carnified. As the whole lung must occupy a certain space in the chest, one portion of it cannot contract without another expanding, and *vice versâ*. It is therefore probable that the two processes of dilatation of the tubes and contraction of the tissue may be in progress at the same time; a bronchitis and blocking of the smaller tubes would prevent an ingress of air into the cells and thus cause a collapse of the tissue, and this might again lead to an expansion of the tubes. It is questionable whether the force of air during inspiration would be sufficient to cause a permanent dilatation without the intervention of the collapsing process, and it is therefore probable, that the two conditions which are so often found together and necessarily associated, a dilatation of the tubes and a shrinking of lung tissue, are in progress at the same time.

Those cases are most difficult to understand where a mass of tubes are immensely distended and in close approximation without a trace of healthy parenchyma between them. In the case of a little girl who had been cyanotic since infancy, and died dropsical, the tubes in the lower lobes reaching to the surface of the lung, were as large as the little finger, whilst nothing but atrophied pulmonary tissue was visible between them. Here there had probably been a bronchitis followed by collapse and subsequent complete atrophy of the tissue. In the case of a woman, without any history of a general bronchitis, the upper lobe consisted of nothing but a congeries of immensely dilated tubes.

Contraction.—From *pressure* without; thus the trachea and bronchi are constantly found compressed and flattened by aneurism of the aorta, and more rarely by other tumours. Mr Wilkinson King wrote a paper in our *Reports* showing that the enlargement of the left auricle, which occurs in some forms of cardiac disease, exerted a pressure on the left bronchus; but we cannot say much in confirmation of the statement, as in the absence of all permanent alteration of shape, found after death in the tube, it is very difficult to prove. Constriction may arise from *disease within*, as from contraction of an ulcer, as you see in these specimens of trachea, and in this one of contracted bronchus, where you will perceive that the tubes are diminished to half their width; the former appearing as if a tight ligature had been placed around them. In all these cases, the patients were *syphilitic*, and there could be no doubt that this was the character of the disease; such, also, has been the case in the few other instances we have seen. Such examples may be known during life, not only by the physical signs of the contraction, but by the very frequent discharge of portions of the cartilaginous rings during coughing.

In a case which occurred more recently the larynx was healthy, but immediately below it a contraction commenced, and extended throughout the trachea, the surface was ulcerated, and was mainly composed of cicatricial tissue; in some places the tracheal rings were exposed, and at one spot was an opening which passed into the aorta; this caused death by hæmorrhage; at the middle portion of the trachea, the internal surfaces actually touched, from the extreme narrowing of the tube. A case is recorded where a syphilitic ulcer of the right bronchus opened into the pulmonary artery, causing a fatal hæmorrhage. Not long ago a man came into the clinical ward with threatening suffocation; tracheotomy was attempted, but the canula could not be thrust into the trachea from its extreme narrowing. In these cases it is probable that the cause was syphilitic, but this need not always be assumed, for it is possible for a peritracheitis to have its origin in other causes.

Congestion and Hyperæmia.—As we have already mentioned, in speaking of other organs, it is very difficult to say where congestion ends and inflammation begins, and this is especially the case in the bronchial tubes: indeed we believe here, as in some other instances, the one passes into the other. This is especially seen in heart disease, where the state of the mucous membrane appears to arise in the first place simply from congestion, but yet in course of time assumes a character which cannot be called by any other name than subacute inflammation. Rarely, except in most acute forms of bronchitis, is the lining membrane of the tubes seen to be so red as in cardiac disease; it is often of the most intense character, and at the same time, the tubes are full of a purulent mucous secretion. A similar condition will be found often in the stomach and intestines, as we shall hereafter mention. It appears as if owing to the tension of the blood-vessels; in heart obstruction, not only is a serum poured out from the serous surfaces, but an analogous mucous secretion from the mucous. This cardiac congestion of the tubes is only an extreme form of what is constantly met with in less degrees in other diseases, where there is obstruction in the circulation, and the lungs are gorged; as, for example, in disease or injury of the spine, productive of paralysis of the chest, the blood may be found almost bursting through the tissue. The congestion may be passive, as in fever and in blood diseases: also in a large number of other cases which we have not time to mention, as in various forms of strangulation and drowning; here the redness is often considerable.

Inflammation of the Air-Passages.—The simplest form is called *catarrhal*, where the mucous membrane is inflamed, and a fluid secretion pours from its surface. It may arise as an idiopathic affection, though it is constantly occurring in connection with other diseases; it is difficult to tell how long it has existed from mere inspection of the tubes, or from the amount of secretion poured out; for this may be very excessive without any great inflammation. The inflammation of the mucous membrane is known, in the first place by the secretion found on its surface; this, if it be of a muco-purulent character, or apparently altogether purulent, shows that the inflammation is violent; but it is rather from the character of the membrane itself that the disease is recognised, for not only is there an exudation from the surface, but also into the membrane, and thus it has a swollen appearance, and the surface is velvety, or granular, besides being highly vascular.

When the *larynx* is affected, the disease is styled *acute catarrhal laryngitis*, the urgency and importance of which is in proportion as the glottis and upper part of the tube is affected; and thus in fatal cases, where an opportunity is presented of seeing the organ, we generally find the glottis of an intense red, and so swollen that it is almost closed, the epiglottis also involved, and the parts below showing ordinary signs of inflammation, with secretion on the surface, and infiltration of the mucous and submucous tissue. The microscope displays an ordinary muco-purulent secretion with epithelial cells. We have seen numerous instances of most intense inflammation of the glottis arise from simple local causes in children who had swallowed boiling water and acrid poisons: in these cases the glottis and epiglottis were intensely inflamed and swollen, and produced suffocation. We would here remind you that suffocation does not necessarily indicate a complete choking of the passage, as the closure is due in part to a spasm arising from the irritation; and perhaps more commonly to a paralysis of the glottis, for it must be remembered that when a muscle is inflamed it is paralysed, and thus the larynx closes during inspiration. Such acute inflammation is often the sequel of chronic disease, as syphilitic laryngitis, &c.; and in some cases, death is produced by closure of the passage, where, on examination, no redness is present, but merely swelling; and this condition is styled *œdema glottidis.* It is often a mere dropsy, or exudation of serum into the cellular structure, and is thus met with in Bright's disease. You will see in these specimens, and very often better still in the post-mortem room, how the glottis is swollen and almost closed by this œdema, and sometimes the epiglottis at the same time.

Another form of laryngitis which would scarcely ever come under the notice of the morbid anatomist is *glandular laryngitis*, where the follicles and glands of the mucous membrane are mainly involved. It is the peculiar affection met with mostly in singers, clergymen, &c.

A much more formidable disease is the erysipelatous or *suppurative laryngitis* in which a purulent secretion rapidly takes place in all the tissues of the larynx and surrounding parts. It is often found in connection with an erysipelas of the neck, when a sore-throat and laryngitis is found accompanying it; in the course of a few days, the patient dies suffocated, with a great swelling of the tissues of the neck. The cellular tissue is found infiltrated with a sero-purulent fluid, the muscles themselves may contain small deposits of pus, suppuration may be found in the muscles of the pharynx, in the tonsils, in the submucous tissue of the larynx, and in the epiglottis itself.

If the inflammation should affect the trachea in an especial manner, the affection would be called *tracheitis;* but we have never seen this tube solely affected; and we shall therefore pass on to the bronchi, and speak of *bronchitis.* This is characterised, also, by the state of the membrane and the secretion from it; as a rule the more purulent the matter thrown out, the more acute is the disease, and this is generally also in proportion as the smaller tubes are involved; and thus we would advise you always to open carefully the ramifications of the bronchi, for you will find more there to account for the symptoms and death than in the larger passages. Thus you will sometimes discover them completely filled with very tenacious purulent mucus, and on making a section of the pulmonary tissue, you will see small drops of thick matter ooze out of the minute tubes: a most important morbid condition, and one you should always look for. At the same time the mucous membrane presents a red, swollen appearance, with its healthy character almost gone—looking, indeed, more like a bleeding fleshy surface; and on section these tubes appear almost closed from the increased thickness of the mucous membrane. If the tubes are distended, as we just now mentioned, the original character of their walls appears quite destroyed, but often they are thickened with a great hypertrophy of the elastic and muscular coats, the longitudinal fibres being very prominent.

Plastic or *croupous inflammation* affects the air-passages, the exudation being membranous instead of muco-purulent. The most important affection of this class is that known under the name of *cynanche* *trachealis*, or croup, a name which appears to denote that the windpipe is alone affected, but in most fatal cases which we examine the whole of the air-passages have been inflamed; but this may be a reason for supposing that, in instances of recovery from croup, so extensive an inflammation could not have existed, and certainly there is no reason to suppose that a *bronchitis* must necessarily accompany an inflammation of the main passages; but we think there is every reason to believe, both from symptoms and necroscopic examination, that the *larynx* is always involved with the trachea. Our opinion is, that in croup the larynx and trachea are always affected, and in more severe cases the bronchi also.

It may be observed that the term croup is not used in conformity with a distinct pathological condition, but as indicative of certain symptoms denoting a closure of the glottis, and, therefore, may include several affections. Thus, besides a spasmodic croup there may exist laryngitis of a simply catarrhal character, and another form where a false membrane is secreted. To both of these the term croup would be applicable during the life of the patient, but it is only to the membranous variety that the term true croup could be used were the condition actually known. Thus it is that the term "croupous" is made to apply to all membranous inflammations of the mucous membranes.

In a case, then, of well-marked croup, you find a membrane covering the larynx and trachea, beginning often on the under surface of the epiglottis, and extending downwards to the bifurcation of the trachea. It has but slight adhesions, and may be removed entire as a hollow tube, as you see in this specimen, which, however, came from an adult. You will perceive how it is placed in real croup in these preparations from children, where it is seen covering the whole surface of the windpipe; in most cases where we have an opportunity of examining the case after death, we find the exudation does not end here, but extends into the bronchi; the membranous character, however, ceases at the bifurcation, and is changed into a corpuscular or purulent secretion. In cases which recover, the bronchial tubes are but slightly, if at all, affected. The cast, when examined, is found to consist of simple fibrillated lymph holding corpuscles in it; the mucous membrane beneath is generally red from vascular injection, but often this stage has passed away, and the membrane is pale. After removal during life, the membrane will often again rapidly form, and if not the mucous surface soon returns to its healthy condition. Sometimes the lymph, instead of forming a complete mould, occurs in patches.

Until late years we have been content to speak of these two forms of inflammation affecting the air-passages, the *catarrhal* and the *croupous*, but now it is necessary to allude to a third styled the *diphtheritic*. You know that since diphtheria has been rife amongst us not only is there observed the more common affection in which the throat is covered with a false membrane, but there is another in which the membrane may extend through the air-passages after the manner of croup. This affection has created a division amongst physicians as to its nature; is it peculiar and a mere manifestation of a constitutional disease, or is it in reality the same as the long known croup? In other words are there two varieties or only one, of membranous inflammation of the air-passages? Is there a purely local affection known as the old-fashioned croup, and is there another of more recent importation associated with a constitutional disease? Or, on the other hand, has the well-known croup been the same disease as diphtheria, but appearing until late years as a milder and more localised affection. The most eminent men in the profession may be found holding the two different views. Those who maintain that there can be a simple membranous inflammation of the mucous membrane apart from diphtheria or any contagious constitutional affection, would refer not only to ordinary croup, but to such a case as occurred here where a child after swallowing boiling water had a croupous inflammation of the larynx, trachea, and bronchi, and to other cases, as of phthisis, where patches of membrane may frequently be found on the interior of the windpipe. It is also maintained that the ordinary croupous membrane differs from the diphtheritic in being thinner, looser, softer, easily separable, leaving the surface healthy whilst the diphtheritic is much tougher, like wash-leather, is a more complete cast, more densely fibrillated and more firmly adherent, and leaving a bleeding surface beneath when removed.

Having spoken of the trachea, we will now pass to the same affection, or *plastic exudation, in the bronchial tubes.* This, as before said, may be merely a part of a general croupous inflammation of the air-passages, but, as a rule, the membrane ends with the trachea, and then gradually passes into a softer or purulent secretion. Sometimes, however, it continues throughout; but, if so, it has been (as far as post-mortem examination is concerned) only when pneumonia was also present, and not in cases which are ordinarily called croup. Thus in the specimen we just now showed you of inflammation being set up by the imbibition of boiling water, the whole passages were involved; but in this case there was pneumonia, and thus we may divide this part of the subject into two,—that where the croup is associated with *pneumonia,* and that where it is *idiopathic.* In certain exceptional cases of pneumonia, the tubes are filled with lymph as well as the lung, as you see in these specimens; the whole lung tissue is blocked up, including not only the air-cells but the tubes themselves, as here exposed. It is a question whether the lymph is formed within the air-passages, or is thrown out from the cells; the slight appearance of inflammation of the tubes has led to the latter opinion, but, at the same time, it is possible for it to be formed in the parts themselves. When the tubes are alone affected, the disease has been called *plastic bronchitis;* this we rarely meet with in the dead body, but we occasionally see it in persons suffering from bronchial complaints, when the casts have been thrown up. These take on the form of the tubes, present an arborescent appearance, and then are expectorated, as you see in this preparation. Instead of the secretion being corpuscular and in the form of purulent mucus, it is lymph moulded into the shape of the air-passages and their minute ramifications. These casts, as well as those associated with pneumonia, may be solid or hollow; the latter being a very common condition, might suggest that they were necessarily thrown out from the adjacent membrane, but we do not think that this constitutes an absolute proof. We have some specimens showing the tubes entirely blocked with lymph in connection with acute pneumonia, and one without the latter complication, but where from the larynx above to the smallest bronchial tube the air-passages are covered with false membrane.

Sometimes, when carefully examined, the casts may be found to be composed of several layers one within the other. The same question arises here as in the similar affection of the larynx and trachea,—whether this plastic bronchitis is merely one variety of mucous inflammation or whether it denotes the existence of a specific or special cause of the nature of diphtheria.

Results of Inflammation, and Chronic Changes.—*Pustules* are met with sometimes in smallpox. We have opened only one body dead from this disease, and this is the larynx from it. The appearances have now much gone off, but pieces of false membrane may still be seen on the surface.

Ulceration.—It is remarkable that simple catarrhal inflammation rarely passes into ulceration, and therefore you must remember that this is not one of the results of bronchitis. The affection lasts for years, but without any breach of surface; and although this may occur to a slight extent at the upper part of the larynx, as the result of various forms of inflammation, yet in the trachea and bronchia simple ulceration is an event rarely witnessed. When it does occur it is associated with tuberculous phthisis in by far the majority of cases. As a rule, then, idiopathic ulceration does not happen; for in the most severe inflammations, when the secretion or lymph is removed, the mucous surface will be found entire, and if found abraded it has generally arisen from the imbibition of an irritating fluid. Occasionally a most extensive ulceration is found with exposure of the cartilaginous rings, but this is generally regarded as syphilitic.

Aphthous.—This is the simplest form of ulceration, and is found associated with the same disease in the mouth. It is characterised by a few very small follicular ulcers being met with in the larynx, and is sometimes seen in phthisis.

Tuberculous Disease and Ulceration is met with in most cases of phthisis that have had any duration. In an early stage minute white points of corpuscular formation may be seen scattered through the mucous membrane; after this a softening takes place, and minute ulcers are formed; at a subsequent period these run together, and, fresh matter being produced, a large ulcer results, as an uneven surface, with white points of deposit on the base. It seems a matter of doubt whether this should be called true tubercle, but some of the larger deposits, as at the posterior part of the vocal cords, appear to be unquestionable tubercle. Sometimes the whole larynx and trachea may be thus affected, but generally the ulceration is in patches; and next to the larynx a favorite place is the trachea, just above the bifurcation, and then not unusually the ulceration extends down into the bronchi and their branches. Its principal seat, however, is the glottis, and the neighbourhood of the vocal cords; and here sometimes disease may be found, whilst all other parts are healthy. An ulcer may be seen at the posterior attachment of the vocal cord, at the base of the arytenoid cartilage; whereby the latter is exposed, and the joint opened. The ulceration then creeps along in the course of the cords until these are wholly destroyed; and in some cases the disease extends along the epiglottis, so that its edges are found eaten away; this produces the most troublesome symptom towards the finale of phthisis, and especially if it should extend still further, as is sometimes the case, into the pharynx; then you find not only the interior but the top of the larynx destroyed, and the outer or pharyngeal surface also involved. Some of you may be asking whether this is the disease known as laryngeal phthisis; we suppose it is, although it is a bad name, as it perpetuates the error that tubercular disease may be located in the larynx alone, as one of the forms of phthisis; probably there is no such disease apart from a similar affection of the lungs themselves.

Syphilitic Disease of the Larynx and Air-Passages.—In the tuberculous disease just mentioned, apart from the small amount of adventitious scrofulous deposit, the affection is characterised by extensive ulceration, whereas in the syphilitic form the peculiarity is the thickening and induration owing to a formation of fibrous tissue. The difficulty is in distinguishing between a syphilitic and a simple inflammatory form of disease; but we believe the majority of cases of *chronic laryngitis* which we meet with are syphilitic, and the more likely is this to be the case when there is a large amount of fibrous deposit present. The disposition, you know, in constitutional syphilis is to the production of lymph, which may subsequently become a tough fibrous tissue; this you see in periosteal nodes, as well as in the same formations in other parts; and thus in the larynx you may find sometimes nothing more than a mass of fibrous tissue developed in the glottis, and almost closing it, as in the specimen now before us; in other cases you find, with this extreme thickening, also the epiglottis thickened and hardened; or this condition may extend down the larynx as far as the trachea; or the whole organ may be indurated throughout, and even sometimes the cellular tissue externally with the adjacent small lymphatic glands all matted together, and implicated in the process. With this induration there is generally more or less destruction of the parts, and in most cases, no doubt, an ulcerative process has accompanied the induration and contraction; and thus the inner surface has either lost its mucous membrane, or presents a cicatriform appearance; the vocal cords may be altogether destroyed, the epiglottis also partly or wholly gone, or presenting a contracted, withered appearance. Sometimes, if the ulceration is considerable, the whole of the inner surface of the larynx presents a shaggy or flocculent aspect, and occasionally the ulceration is continuous over the glottis, with an ulcer of the pharynx; in such a case the question may arise as to the original site of the disease; but, as both these parts may be independently affected, it is possible that the disease has progressed in both parts simultaneously. The earlier stages of syphilitic inflammation of the larynx, would seldom be met with on the post-mortem table; but during life we find at the outset of constitutional symptoms a simple redness with secretion as in the catarrhal form; to be followed, perhaps, by raised patches on the mucous surface, which are sometimes designated condylomata.

Since special attention has been given to the internal syphilitic affections, the peculiarities of the more chronic laryngeal diseases have been observed, and a real gummous perichondritis of trachea and laryngeal cartilage, has been several times noticed. A homogeneous material may be found surrounding the air-passages, and involving their coats, and spreading around the œsophagus and neighbouring tissues; we have seen the œsophagus laid open, and sometimes, by the softening of the syphilitic material, an external abscess, and necrosis of cartilage. The mucous membrane is thickened, puckered, or ulcerated.

Other parts of the air-passages may be affected as well as the larynx, as you see in this specimen, where the lower part of *trachea* is very much thickened, and its surface ulcerated; and in the preparation we just now showed you, of contracted *bronchus* arising from an ulcer, the nature of the disease was clear, in the fact of the patient dying of syphilitic laryngitis; the contracted trachea also had the same origin. As we before mentioned, in some of these cases of ulceration of the trachea the rings are laid bare, as you will see in these specimens;

and which sometimes become detached during life, if the patient recovers.

A Cancerous Ulceration, or destruction of the upper part of glottis and epiglottis, in connection with the same disease in the pharynx, sometimes happens, as you see in these preparations, though it is not very common.

Typhoid Ulceration.—One of the concomitants of typhoid disease is an affection of the larynx. It is very rare in this country, though it sometimes occurs, and is generally described by continental physicians under the name of *laryngitis typhosa.* This larynx, however, with this typhoid intestine, shows the affection which is described. At the posterior part of the larynx, at the junction of the vocal cords, you see a little hollow space on each side ; this cavity was produced by the softening and sloughing of a deposit which had occurred at this spot. The remarkable circumstance in the case was, that this cavity led into a space between the trachea and œsophagus, through which air had escaped, and had given rise to general emphysema. We may here remark, that emphysema has been occasionally observed in the course of typhoid fever, and in this affection probably may lie the explanation.

Disease of the Cartilages.—We have just now stated that very many of the chronic laryngeal affections have a syphilitic origin, and wherever the induration and thickening exists of which we have spoken, there is considerable suspicion of this being the case ; there appears, however, to be an affection of the laryngeal cartilages independent of the specific taint. In many instances, such as in the case from which this specimen came, there was no syphilitic history. The disease advances with considerable pain and swelling about the larynx, accompanied by suppuration either within or without. After the abscess is opened, the cartilage is seen to be dead and of a black colour, and like a piece of leather ; very commonly, however, the portion of diseased structure, as one ala of the thyroid, is found converted into bone, which becomes exposed and exfoliates, if death do not previously ensue. The nature of the pathological affection is not altogether clear. When the inflammation has begun on the surface, the name *perichondritis* has been given, but, as in many cases of the disease an ossification of the cartilage has occurred, it has been thought that this is the commencement of the morbid condition, and that necrosis follows, attended by suppuration and exfoliation ; whilst some think, that during the slow inflammatory process, whether arising in the first place from syphilis, injury, or other cause, the change in the cartilage takes place. This affection of the cartilages is to be considered independently of the chronic laryngitis, where the mucous membrane is especially diseased, for it is accompanied by different symptoms, and runs a different course. Sometimes an abscess opens externally, and the cartilage is exposed, as you see here; or the abscess may pass up and open into the pharynx, as in this specimen. In this you see it is the cricoid which is especially affected, and a fistulous opening leading from it to without. One specimen refers to the case of a child who died of laryngeal abscess with necrosis of the cricoid following measles.

We may here refer you to several specimens, of which the pathology is very obscure, where an opening exists between the trachea and the œsophagus; in these cancer was suspected, but none was found after death; and, if due to simple suppuration, its cause was not evident. More rarely the hyoid bone becomes necrosed, giving rise to abscess and exfoliation.

Ossification.—This sometimes occurs in various parts of the air-passages. We have already said that in cases of disease of the cartilages, attended with suppuration, it is very frequently met with. In old persons the larynx is thus found ossified, as you see in this specimen; also the rings of trachea and bronchi may be ossified. And here you see bony deposits forming plates and irregular patches all along the trachea and bronchi; these are quite independent of the rings, and are formed in the mucous membrane; the microscope showing true ossific structure.

Adventitious Growths.—These occur mostly at the upper part of the larynx, and are of the polypoid character. The favorite seat is on or near the vocal cords; the simplest and smallest of these growths are of a *warty* or papillomatous kind, as you see in this specimen. When placed under water, you see shaggy or villous processes float out, and they are then seen to be attached to the vocal cord. Sometimes a growth of this kind reaches a larger size, and nearly fills the glottis, as in this one. They then constitute tumours, and may be really cancers of the epithelial variety, or *epithelioma;* such a one you see here, which was removed during life, by Sir A. Cooper; and only a short time ago Mr Cock had a similar case, but there the tumour grew from the external surface, and protruded into the pharynx. Some such tumours are of a simple fibro-cellular character, and should rather be called *polypi*. None of these circumscribed or pedunculated tumours are truly cancerous, a disease disposed to infiltrate and destroy; but this specimen appears to be true *cancer,* as it is involving all the upper part of the larynx, and protrudes both externally and internally. Primary true cancer is rare; it is generally of the epithelial variety, which spreads and destroys all the superficial parts, but the disease is local. Cancer may affect the air-passages from without, and thus in cancer of the œsophagus the disease may extend to the trachea, though the latter is rather destroyed by it than involved in the disease; in cases of cancer of the root of the lung, the disease often involves and penetrates the tubes, as you see in this preparation. In some cases it has involved the œsophagus, and more rarely sets up an inflammatory process causing external abscess.

Injuries.—Injuries from direct violence are very rare, but those most usually seen are in cut throat. Mostly, however, the instrument of destruction passes between the cartilages, or rather above the thyroid, and so they are uninjured; if they should be incised, and union take place, it is, as we have before said, not by a reproduction of cartilage, but by fibrous tissue, which, in the course of time, may become bony.

Foreign Bodies.—This is a subject very important in surgical practice, as you may often be called in to perform tracheotomy on account of extraneous substances having found an entrance into the windpipe. You will see among our preparations, coins and pieces of bone which have thus passed in. During life the substance may be found constantly moving up and down the trachea, endeavouring to make its exit, and first passing into one bronchus and then into another; when, however, it has become permanently fixed, and death has been the consequence, it has, we believe, been invariably found in the *right* bronchus, or one of its branches, where it has produced a local inflammation, abscess, and destruction of the lung tissue.

Sputa.—This is scarcely a subject for these lectures; but we would advise you to familiarise yourselves with the different appearances of the matters expectorated, as they are seen by the naked eye and by the microscope—such as the frothy mucus of bronchitis, especially where there is much difficulty in expectoration; the large quantities of uniform purulent matter in acute bronchitis; its nummular character in chronic disease of the tubes and in phthisis, and the rusty expectoration of pneumonia; also the black spit in some forms of chronic broncho-pneumonia, or the large quantities of matter (which is often fetid) from an empyema; then the application of the microscope, which displays in all these, as a principal ingredient, granule or pus cells; the dark specks due to larger cells containing black points of pigment; the red expectoration showing blood globules, and with these epithelial cells, and round nucleated pavement cells from the smaller bronchial tubes or perhaps pulmonary vesicles; ciliated cylindrical epithelium from the larger tubes, and with these may be associated larger epithelial cells from the mouth. In cases of phthisis, where the lung is disorganised, portions of the tissue may sometimes be found; and it has been stated by those who have made a long study of the subject, that tuberculous matter may be recognised by the irregular form of the cells.

PLEURA

Inflammation.—Pleuritis presents various conditions, according as it is acute or chronic, and according to the kind of material effused. Although, as before said, acute disease may pass into chronic, yet, very often, the affections are so different that they do not so much resemble each other as some diseases bearing distinct names; a fluid effusion, for example, on the one hand, and a thickened pleura, approaching almost in character to a new growth, on the other, have very little in common.

It is very possible that pleurisy may arise under two very different pathological conditions according as the pulmonary or the costal pleura is affected. In the former case it may be associated with pneumonia, or its cause may be coextensive with that which has produced the inflammation of the lungs; in the latter it may be of a purely local character. There may be, indeed, the same distinction as we draw between arachnitis having its cause from without in connection with the dura mater, and that which originates essentially from within in connection with the brain itself.

An acute Pleurisy, if seen a day or two after its first onset, is characterised by an effusion of lymph forming thin films over the surface of the pleura, and causing the lung to adhere to the chest. This thin layer can be easily peeled off the serous membrane, which is then seen to be of a dull hue, and at the same time highly vascular. The hyperæmic condition is best witnessed on the costal pleura, where the membrane is often intensely red; but on the lung, as it is frequently associated with pneumonia, the pleura presents a dull and whitish aspect. Even earlier conditions of inflammation are sometimes seen, where from a dull-looking pleura a very minute quantity of exudation may be scraped off; but generally much more effusion is seen than has been mentioned. Thus the whole lung may be covered with a layer of soft lymph completely hiding it, and the costal pleura be covered in like manner: and in such a case the lobes would be found adherent together, and the base of the lung to the diaphragm. We cannot say in what proportion these different parts are likely to be affected, but interlobar pleurisy, and that of the base of the lung, are probably as frequent as any. Combined with this *effusion of lymph* there may be some *serum*, which will collect in the meshes of the fibrinous exudation, and may be found in considerable quantity in the pleural space. Local patches of pleurisy may be found over the consolidated portions of lobular pneumonia, apoplexy of the lung, &c. After a moderate amount of lymph is thrown out, some absorption takes place, and the remainder forms into a fibrous tissue which causes the lung to adhere to the chest. If you examine one of these adhesions by the microscope, you will find that it presents the appearance seen in this sketch, being composed of fibres, the surface covered with pavement epithelium, and the black specks in it consisting of pigment cells, as in the colouring matter of the lung.

Sometimes, and owing generally to a constitutional cause, the effusion becomes purulent, and an *empyema* is formed. The lymph which is thrown out forms a layer over the pleura both of chest and lung, like the wall of an abscess, and the matter is contained within. If the chest is filled, the lung necessarily becomes compressed, and, if not adherent, contracts close to the spine. It is doubtful whether this purulent matter ever becomes absorbed, and therefore it either leads to the death of the patient, or attempts to make its way out; unless, indeed, it be in small quantity, or circumscribed, when it possibly may remain inert for a considerable period. Not long ago, we had an opportunity of examining the body of a woman who had a local collection of pus, or an abscess, at the very bottom of the chest, between the lung, diaphragm, and ribs; this was shut in, and probably had existed for some years; for during this time there had been physical signs of fluid at the part, and a history of pleurisy three years before, and we think we have met with other similar instances. If the abscess in the pleura attempt to make its way out, it is generally either externally through the thoracic parietes, or internally through the lungs. In the first case, it is remarkable that the favourite site for its exit is not at the spot most favorable for tapping; that is, posteriorly, at the seventh or eighth rib, where we generally insert a trocar; but nature mostly evacuates the contents anteriorly and higher up. Thus you may observe a protrusion commencing as high as the third or fourth rib in front; after a time the abscess bursts, and the pus continues to discharge for many weeks through a fistula, during which time the lung may again expand, but generally the chest falls in, so that during the process of cure, considerable contraction of that side of the thorax is occurring, accompanied sometimes by a slight curvature of the spine. If the matter make its way *through the lung*, it opens into a bronchial tube, is spit up, and is so got rid of; if the walls of the abscess should contract as the matter is diminished in quantity, no air need enter the chest; but sometimes as the fluid escapes, some air enters, and thus decomposition takes place; the purulent matter becoming very fetid. We have, then, in the chest, fluid mixed with air; in fact, a *hydro-pneumo-thorax*. As in such a case, however, when you percuss the body, it is possible you may not elicit the tympanitic sounds which air should give, we may explain how it differs from the more usual form of pneumo-thorax which we commonly meet with. In most cases, this affection arises from a rupture of a diseased lung, and air escaping into the chest produces, in consequence, a tympanitic sound on percussion; subsequently, too, from the pleuritic effusion which is sure to arise, a splash is heard when the body is shaken. In the case under consideration, although fluid and air may occur together in the chest, yet, from the fact of the abscess being circumscribed and surrounded by dense walls, that vibration of the ribs which is necessary to elicit a resonant note is wanting, although, if the space be large, a tympanitic sound may be produced from the deeper parts by a more violent stroke of the fingers. We mention these particulars because, as we have told you always to percuss a body before examination, you may be surprised to find a hydro-pneumo-thorax, and yet only a dull sound elicited on slight percussion with the fingers.

As *pneumo-thorax* is a distinct pathological condition, it requires a description of its own, but, as we are on the subject, we will complete our remarks upon it. Air in the chest arises either from its escape from the lung or its entrance through the thoracic parietes. It is remarkable, however, how rarely the affection occurs from *injury to the walls of chest*, the opening being closed by elasticity of the skin, or adhesions of the lungs; and thus you may remember, in the case of the police-officer who was shot through the chest, no air got into it at first; and it was only as the wound sloughed, and in the course of some days, that this occurred. In most cases, pneumo-thorax arises from escape of air from the *lung* into the chest, either from *disease* or *injury;* if the latter case, it is mostly from fracture of the ribs, and this is often associated with emphysema, or diffusion of air into the cellular tissue of the skin. Under these circumstances when the chest is opened, the lung is found contracted, and if death has been rapid there is no fluid except some blood from the lacerated lung; if death be not immediate, then some inflammatory effusions are present as well as air. Fracture of the ribs is not necessary to produce a laceration of the lung; for in the case of a child run over by a cart, the lung was torn, without any injury to the parietes; and another remarkable circumstance in connection with injury of the chest is, where, after a penetrating wound, instead of pneumo-thorax taking place, the lung protrudes, constituting a hernia of the lung. We believe we have seen two cases of pneumothorax arise from tracheotomy, and we mention the circumstance because we are not aware that it has ever been alluded to: in one case where, after tracheotomy, death occurred without sufficient reason, both the lungs were found contracted in the chest, and the cellular tissue in the posterior mediastinum was filled with air, producing large bubbles, which we think, had burst through the pleura into the chest. In another case, where most extensive superficial emphysema followed the operation, the breathing became laborious before death, and the lungs were found contracted in the same manner; the emphysema having penetrated the mediastina. Most cases of pneumo-thorax we meet with, arise from *disease*, especially phthisis; and this being one cause of sudden death, may have come on after the last visit to your patient; you should, therefore, always percuss the body before opening it, and, if suspecting it, you may first put a trocar into the chest, when a gush of air escapes, which is audible, or will blow out the flame of a lamp placed near the opening. Most frequently, being not immediately fatal, a pleuritic effusion has occurred, and the case is one rather of hydro-pneumo-thorax, and thus, when the chest is opened, the lung will be found compressed, but not into a very small space, from its being diseased; the remainder of the chest, as the outer and lower part, will be found partly full of purulent fluid; if you now place a tube in the trachea, and blow by the mouth, or by means of our bellows apparatus, you will detect the opening in the lung by the bubbles of air rising through the fluid; or, should the opening be above the level of the fluid, the air may be observed escaping; if not, the remainder of the chest may be filled with water, and the experiment be again made. On examining the lung, the opening will be found to be caused generally by the bursting of a small phthisical abscess in a very early period of pulmonary disease and, this already communicating with a bronchial tube by a previous ulcerative process the direct passage will be readily found between the main bronchus and the opening. The latter will generally be met with about the middle of the lung; and in the majority of the cases which we have examined it has been at the lower edge of the upper lobe; in one case, it was at the lower surface of the upper lobe. In speaking of the first form of this disease, where the primary affection is an empyema which makes its way into the lung, the cases are not sufficiently common on the post-mortem table to warrant a positive opinion as to how the opening of communication is formed, and what is its favourite site. In the phthisical form, the opening, remember, precedes effusion. As the empyema may be circumscribed, we apprehend it chooses the nearest spot of lung through which to penetrate; but in one fatal case we examined, after a most rigid examination, we failed to find any perforation, excepting, indeed, a number of small openings which were seen after removal of the lymph in the softened lung tissue. Such a case may, perhaps, warrant an opinion which was held by Dr. Barlow, that the matter may be taken up by the surface of the lung, and discharged by the bronchial tubes, without any direct opening.

Chronic Pleuritis.—This may be essentially chronic, or result from an acute attack, and may be attended with various results, as effusion of fluid, serum, or pus, or a mere thickening of the serous membrane. So difficult is it to say in some cases where acute inflammation ends and chronic begins, that we have spoken of empyema already, as the case where pus is poured out under acute affections, but very often it is essentially chronic. So, also, with effusions of serum; in some forms of pleurisy a very rapid pouring out of fluid takes place, not so quickly, indeed, as the more solid products of lymph, which occur in a few days, but generally in the course of about two or three weeks, when the chest may be found having some lymph lining it, but at the same time filled with a serous fluid. In cases where the exudation is wholly serous, the disease is essentially chronic, and is so slow in its progress that its presence would be unknown were it not for the physicial signs. In these cases the chest is filled with pure serum, and the lung is compressed against the spine; generally the fluid is again absorbed, but if by chance an opportunity for examination should occur, the pleura presents no difference from a healthy membrane, and thus this *chronic pleuritic effusion* is like other serous exudations whose pathology is unknown, as, for example, hydrocele; we know, indeed, that the serous membrane is not in a healthy state, or the increase of secretion would not take place, but the deviation from the normal condition is seldom appreciable to the eye. If the lung has been compressed for some time, it becomes permanently carnified and finally the whole tissue atrophies.

As a chronic inflammation may result in effusion, so it may produce a mere *thickening of the pleura*. In one case this may have been preceded by an acute attack, and in another the increase of thickness is so slow in its progress, that the change is more like a growth than a product of inflammation. In the former, the process from beginning to end may be understood on carefully examining the chest; thus, if death occur some weeks or months after the first attack, you may find that the lung separates with tolerable ease from the thorax, the lymph on the surface being still soft; but on making a transverse section through the lung, the layer below will be seen more dense, and the new tissue below that, still more so, until we arrive at that next the lung, which is the hardest of all, and incorporated with the pleura. The earlier layers are thus seen to be the hardest, and the recent ones the softest; this we call a *chronic* process: not one synonymous with *old*, or one that is altogether past, but one which commenced at some antecedent period, and continued up to the time of examination. Another chronic process, but still slower, is where there is no evidence that at any time a soft material has been effused, the increase of thickness being imperceptible, and more allied to a growth; it is a question with some, whether such a thickening *must* not have been preceded by a softer effusion of lymph; but from the observation of several cases where there has been no history of any acute action having taken place, we are quite of the opinion that this is not necessary, and that such thickened pleura need never have presented any other appearance as regards its texture, than that actually found at the termination of the case. In these instances you see the lung closely adherent to the chest, by tough tissue as hard as cartilage, so that the knife is required to cut it out; in one man, we remember, this new tissue was an inch in thickness, and was associated, as is usually the case in these severe forms, with cirrhosis of the lung; the whole organ being sometimes affected, at other times merely a part, and the more frequent place the upper lobe. The question here alluded to, of the mode of formation of such a tissue, is one still open and discussed by pathologists; the point being whether a preliminary stage of effused lymph is necessary for its production, or whether the process is not rather allied to a slow growth; that is, whether an exudation, which is at first separate, subsequently becomes organised and attached to the original surface; or whether the new tissue has ever been separate from the latter, or has rather been an offshoot from it. We think, that both occur, and that one does not preclude the other; it is true, no doubt, that an effused lymph may subsequently become organised and form a part of the original tissue from which it proceeded, but at the same time, that the other is none the less true—that is, that the increase to the surface is so slow and to so slight an extent, that no actual separation from the original membrane could ever have been witnessed; it may commence, however, in the form of elementary cells, as in a positive exudation, for even in well-formed new growths of a fibrous character, which go by the name of tumours, the nucleated fibres are preceded in most cases by a production of such cells.

Morbid Growths.—We have already said, that in cases of cirrhosis of the lung, where the pleura is excessively thickened and indurated, the process is allied to a growth. Sometimes, in such cases, an earthy deposit takes place, or an ossification, as it is called, in the new tissue, and a large *plate of bone* the size of the hand may be found on the surface of the lung, or in some cases at the base of the lung, where the latter adheres to the diaphragm. Such large bony pieces you may see in these specimens. You will hear such plates called ossifications, as also the thickening of the pleura styled cartilaginous; but you must remember that all that is meant by such expressions as cartilage-like, and bone-like, is, that in the one case the tissue is simply fibrous, and in the other, consisting of a matrix of simple fibre containing earthy matter, or salts of lime contained in the meshes of the inflammatory product.

Fat.—A few instances have been recorded where a layer of fat has been found covering the lung.

Bone and *cartilage* may, however, occur in the lung, and therefore, on the pleura, but if so, generally as secondary deposits to similar growths in the external parts of the body, and very rarely as primary; so rarely, that in the one or two cases where we have seen them, it is a question whether, if the whole body had been carefully examined, some similar growths might not have been found elsewhere. In a case not long ago examined, we found small bony plates on the pulmonary pleura, and these, when viewed by the microscope, were found to be composed of true osseous tissue. This tumour, found in the pleural cavity, is said to be fibro-cartilage, but we have not examined it.

Cancer.—When cancer involves the lung it may also attack the pleura, and it generally occurs as a secondary deposit; in these cases it is scattered all over the pleura, and sometimes without the lung itself being involved. It is generally seen in the form of white flat and smooth patches, scattered all over the pulmonary surface, but penetrating occasionally into the lung tissue. These are generally very hard, being composed of fibrous structure as well as of cells, and if only in small quantity, not easily recognised, or the idea of cancer not suggested, unless this is seen also to prevail in the abdomen, or elsewhere. The costal pleura may be similarly affected, and sometimes severely, by large vascular medullary growths springing from its surface, or occasionally by cancer actually growing through the thoracic walls, from the exterior, as in carcinoma of the breast.

Tubercle.—In cases of general tuberculous infiltration of the lungs, the pleura is frequently similarly affected, and thus the whole surface may be found covered with small miliary tubercles. There is also another form of more acute deposition, which might also be called scrofulous pleuritis, where in the case of an inflammation in a scrofulous individual, an effusion takes place in a subacute degree, but of a tuberculous character. Such an instance you all had an opportunity of seeing, only lately, in a boy, who, after receiving a blow on the chest, had a pleurisy set up; and at time of death, three or four weeks afterwards, the lung, when torn from the thorax, was found to have a yellow scrofulous matter effused upon it, or a lymph putting on the tuberculous character.

Amongst the morbid contents of the chest we have mentioned *air*, *serum*, and *blood*, and these have already been alluded to under several heads. We have said *air* may get in from without through the chest, in case of an opening in the parietes, but more usually it arises from the lung, and this occurs either from injury or disease; in the first place, mostly from a fractured rib lacerating the organ, or its being punctured by the stab of a weapon, or bullet. It is remarkable that the latter does not always cause pneumo-thorax, even if the organ be considerably injured. If from disease, it is generally from rupture of the lung occurring in phthisis. Dr Hughes relates a case where he thinks air escaped from an emphysematous bubble; but, recovery taking place, there was no opportunity of verifying the diagnosis.

Serum.—We have already alluded to this as one of the products of inflammation, but it may also be a passive effusion. It is thus found associated with dropsy in other parts of the body, in connection with heart or kidney disease. It is often difficult, however, to know why it should occur on one side rather than the other; but in Bright's disease of the kidney you may often find a very simple explanation in the fact of one lung being adherent, and, there being no space for the effusion, the lung itself being œdematous, while the effusion occurs on the other side where there are no adhesions, and the lung is consequently compressed. If it be true, according to the statement we have already alluded to, that the left bronchus is compressed by the left auricle in some forms of heart disease, it is easy to conceive how the pulmonary vessels would also be constricted, and thus effusion might rather be determined on the left side. We mention this, as it is a point yet to be worked out. It may arise altogether from a local cause, as when pressure occurs on the pulmonary vessels, as in cancer of the bronchial glands, and thus is a very common termination to cases of carcinoma mammæ. This passive effusion is called *hydro-thorax.*

Blood.—Blood in the pleural cavity may have many sources; thus, a rupture of an aneurism may cause it, or a wound of the lung from fractured ribs. In such a case, a short time ago, where death occurred after some days from pleurisy, lymph was found mixed with blood, forming a layer on the surface. It may also, occasionally, arise, as we have a few times seen, from laceration of the liver, the lower ribs being fractured. The diaphragm and liver have been torn, and blood has been effused into the chest. This, of course, is when the right side is injured; a similar injury on the left side, with laceration of diaphragm, would tend to diaphragmatic hernia, or displacement of the stomach into the chest. In all such cases the source of blood is manifest; but there is one case where students are often considerably puzzled, and that is, where blood is mixed with inflammatory exudation, as in *hæmorrhagic effusion.* We have already alluded to a similar state in the pericardium: it arises probably from some constitutional cause, where there is a tendency to purpura, and thus, after the pleurisy has existed for some days, blood is poured out. On opening the chest, the lungs seem compressed, and the cavity filled apparently with blood; but on careful examination the serous membrane will be seen to be covered with lymph, and in its meshes blood is effused. Whether the source of this is the original tissue softened by the inflammation, or the rupture of new-formed vessels, has not yet been satisfactorily determined. In cases of *injury to the chest,* from stabbing or gunshot wounds, death often ensues from a pleurisy in which inflammatory products are found mixed with decomposed blood, the source of blood having been either a wounded intercostal vessel or wounded lung.

Contents of the stomach may sometimes be found in the chest, and we tell you of it because it may puzzle you if met with; it is not common, we have seen it two or three times. You know that the stomach becomes dissolved sometimes by its own juice, and the contents escape into the abdomen; in the same way, from the body lying on its back, some of the gastric juice passes into the œsophagus, and, there causing a similar solution, the contents may escape into the chest, and then, indeed, begin to act on the lung, softening and dissolving its structure. It is very important, sometimes, to recognise such post-mortem changes, and distinguish them from ulcerations; for it was in a case of poisoning by arsenic, that one of the cases of digestive solution of the œsophagus occurred.

We have already alluded to the case of diaphragmatic hernia, where the stomach, or transverse colon, may pass through a ruptured diaphragm into the chest.

LUNGS

Hypertrophy.—We know very little of hypertrophy of the parenchymatous organs, as we shall presently have to say with reference to the abdominal viscera; that is, in an absolute sense, although a relative hypertrophy is common enough, the wasting of one part (or of one organ when it is double) being compensated for by the enlargement of another; and thus, as regards the lungs, when one of them has been long atrophied the other becomes larger, and thus it is seen not only filling its own side, but crossing the median line and heart and reaching to the opposite side. We would not, however, positively assert in such a case that the lung is actually hypertrophied, because enlarged; but, judging from the analogous case of the kidney, we see no objection against its being so.

Atrophy arises from various causes. The most simple case is where the lung has been long compressed from pleuritic effusion or thickening, when the tissue has eventually undergone a structural change,—in fact, become atrophied. The same may occur in a part of the lung, as presently to be mentioned, or a lobe may be so affected by chronic pneumonia as to waste away.

Condensation of the Lung—Atelectasis, Apneumatosis, &c.—This is the condition of a lung while in the fœtal state, or while in utero, which has never yet contained air. The cells are collapsed, and thus it constitutes a solid mass, which sinks in water, like any other organ. After the lung has been used, if the air be squeezed out the same state is again produced; this is well seen in pleuritic effusion, where the lung becomes compressed against the spine, and when taken out is quite airless, and sinks in water; this is at once distinguished from a hepatized lung, which also sinks in water, by being tough and resembling a fleshy substance; hence the name *carnification*. The air in this case is forced out by external pressure, but the same thing may occur in parts of the lung from mere want of power in the respiratory process to overcome the natural elasticity of the tissue, and, consequently, certain lobules, or larger portions of the lung, may return to the fœtal state, and, inasmuch as this resembles the same condition where, from debility of the child, the lung has never been thoroughly filled, the name *atelectasis*, signifying *imperfect expansion*, has been given to it. This is congenital atelectasis.

In infants who survive their birth but a few days, the lungs may be thus found imperfectly expanded, portions being quite airless, dense, and sinking in water. At a later period of life, the same state may still be found, and then it is very probable that the lung may have once been wholly expanded, but, owing to some weakness of the patient, portions have again resumed their original fœtal condition. If, indeed, this does not occur from mere inability of inflation, it has of late been very satisfactorily proved to arise from any cause which may obstruct the entrance of air into the lung. We believe the first person who directed attention to this return to the fœtal state in certain portions of the lung was Dr Alderson, who, several years ago, pointed out how this condition is often found in conjunction with hooping-cough, and that it had hitherto been looked upon as lobular pneumonia. Dr Bright also had observed the same fact.* It may, however, follow ordinary bronchitis, especially when occurring in children. The lung, which in a child is of a light colour, is seen to have a number of dark red portions on its surface and towards the periphery wedge-shaped masses; these were formerly supposed to be hepatized parts; they are more solid than the healthy tissue, and often sink when placed in water, but, unlike a hepatized lung, the surface is not granular when a section is made, and, moreover, these parts are depressed below the surface, whereas a mass of inflamed lobules would be raised. This is, of course, what you might expect, when in the one case the cells are filled with an albuminous matter, and in the other they are simply collapsed or airless; they are both increased in specific gravity, but one is enlarged, granular, soft, and exudes a fluid on pressure, while the other is diminished, smooth, tough, and dry. The proof we have of this being its true condition is in the power we have of artificially inflating these portions, and thus reducing the lung to its original state. We should say, however, that the two conditions may be combined; that is, an inflammatory exudation may occur in the collapsed part, for when the collapse occurs from the plugging of a bronchiole an extreme congestion naturally occurs. The collapsed portion is red, subsequently may become œdematous, and, finally, may be the seat of inflammatory changes. It is a state especially met with in children, but also in old people, particularly in the bronchitic, where a collapse of a large portion of the lung may occur, especially at the posterior part, and accompanied by recent inflammatory effusion; thus, portions of the organ are soft, easily broken down, and exuding serum, while others appear simply airless, but are firm and dry. A more than usual interest is attached to this subject, since it has been made the foundation for a new theory of emphysema, as we shall presently mention.

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* Dr Bright, in his 'Medical Reports,' published in 1828, has the following:—"I had the opportunity some time ago of examining the chests of two children who died of hooping-cough. In both the breathing had become more and more laborious until death. There was not a vestige of what we would ascribe to pneumonic inflammation, but the greater part of the lung was in a state of decided emphysema, while many of the lobules about the edges of the lungs were flattened as if they had not admitted the ingress of air for a considerable time. The bronchial tubes were a good deal loaded with viscid matter.

Emphysema is divided into two forms, the *vesicular* and the *interstitial*. The first is the most common, and that which is generally implied when the term emphysema is used, although it so happens that it is to this very case the term is not strictly applicable, for, as used elsewhere, it means an infiltration of air into the tissue, which, in the case of the lung, is a comparatively rare occurrence.

Interstitial or *interlobular emphysema* is the more rare form, and is due to the escape of air beneath the pleura and into the tissue. It is generally recognisable by a number of air-bubbles beneath the pulmonic serous membrane, appearing like a number of beads scattered on the surface. It is mostly found in the lungs of children who have long been subject to bronchial affection, as hooping-cough, and in these you will see, as in this specimen, the surface of the lobe covered by a number of air-bubbles.

The *vesicular* or *ordinary emphysema* is an affection which has of late attracted much attention, from the novel theories respecting its mode of formation, and, therefore, we will venture upon a few particulars concerning it. Emphysema, when existing to any extent, is at once recognised, and we are constantly showing it you in old bronchitic cases, and a specimen of it you see here. The lung appears to have a number of additional pieces or bladders attached to its surface, and some of them are pedunculated. They are seldom seen except along the front edge of the lung. When cut through, you perceive they are composed of a highly rarified tissue, like sponge, or the lungs of a reptile, consisting of large cells or spaces, which are either immensely dilated pulmonary vesicles, or several vesicles which have run together from the rupture of their partitions. It is mostly in old cases of bronchitis that you will meet with this condition, but not uncommonly, also, in chronic phthisis. The connection of emphysema and these diseases has always been recognised, and as consequent upon them. Bronchitis is evidently a very usual cause, and thus Laennec, who was almost the first to study diseases of the lungs by a philosophic method, framed an explanation which has generally been received up to the present day. He thought a bronchial tube became obstructed by mucus, and consequently, the air in the pulmonary tissue leading from it could not escape during the expiratory process, and therefore, from the great compressing force upon this part of the lung, the air distended the vesicles or burst through the tissue. Although such a process could not be absolutely proved, and there were many difficulties in the way of explanation, yet the evident connection of emphysema with bronchitis, and the many reasons to believe that it occurred during violent fits of coughing—that is, in the expiratory process—caused most physicians to follow Laennec, and, consequently, his doctrines were for many years taught in this theatre, even in spite of the promulgation of the more recent theories. The latter theories have resulted from the discoveries made with respect to collapse of the lung, or atelectasis, as we just now explained, and for their full development we are mainly indebted to Dr Gairdner. It is said, in the first place, in opposition to Laennec, that any obstruction of a bronchial tube could not cause the retention of air behind it, but rather the opposite condition, for any plug of mucus in a conical tube would act as a valve, and thus, although not admitting air, it would easily allow the egress of that already in the vesicles; and to prove this, experiments have been instituted on the lower animals, in which bullets were placed in the bronchial tubes, when the tissue to which these led, so far from becoming expanded with air, became collapsed; the ball, acting as a valve, allowing air to escape, but not to enter. This immediately refuted Laennec's theory, for, so far from emphysema being produced, the lung contracted and became dense, in the manner already mentioned. Another objection, too, given is, that a pressure on a part of the lung during the expiratory process could not cause it to dilate, for equable pressure on a membrane full of air, however delicate, cannot alter its form; again, if the disease were produced after Laennec's idea, those parts of the lung should be found emphysematous where the tubes are most liable to obstruction; but the opposite is the case, the one occurring at the anterior parts, and the other at the posterior. With these objections, Dr Gairdner has expressed his opinion that emphysema occurs not during expiration, but during inspiration; and that, although the first step in the process is, in most cases, a bronchitis, this is only effectual in producing a collapse of a portion of the lung, which is the condition immediately antecedent to the dilatation of another part of the tissue; the latter, indeed, being compensatory to the previous contraction. He thinks a tube, and this is generally one leading to the posterior part of the lung, becomes obstructed; that the portion of lung to which it leads, collapses; and that, consequently, during inspiration other portions, generally the anterior, become distended, to compensate for the proportional decrease; that the chest, indeed, when it expands, must be filled by a certain bulk of lung, and that, consequently, when one part of the tissue has become contracted, another distends. Dr Gairdner does not say, as some have misunderstood him, that when a part of the lung is functionally obstructed, as in pneumonia, another part expands to make up for its loss, but simply that, when one part is contracted, another expands to supplement its bulk in the chest; again, if the tissue of the lung should contract from any other cause except bronchitis, emphysema would occur, and thus its existence in very chronic cases of phthisis, where portions of the tissue are found puckered up by tubercular or inflammatory deposit. This theory was readily received by many, although, as before said, several of our older and most experienced physicians still held by Laennec; and declared that, although there might still be difficulties in the old views, yet they could not but think that emphysema occurred during expiration. Subsequently Sir W. Jenner advocated this view, as opposed to Gairdner's, and maintained that it is during the expiratory process that the disease really occurs. He does not believe, with Laennec, that it is due to a compression of the air behind an obstructed bronchial tube, for the objections already stated are, no doubt, insuperable; but he considers Gairdner's simile of equable pressure on a bladder of air not true of the lungs, for the pressure of the chest is not equal; the lower portions of the lungs may have an equable pressure exerted on them by the posterior part of the chest together with the lateral part and floor, but the anterior part of these organs towards the mediastinum and the apices beneath the clavicles have not the same force exerted on them, and consequently if the whole lung were more than usually pressed by the thorax, these latter portions would endeavour to protrude and become distended. The expiratory force, therefore, is quite equal to the production of emphysema in those parts, where the pressure is less, and in cases of violent cough, the apex of the lung may be actually seen forced up above the clavicle; moreover, this condition may occur without any appearance of compensatory contraction in any other part. In the case of M. Groux, who had the fissured sternum, the anterior part of the lung bulged through the opening during the act of coughing. And if the example of draught animals be taken, we find emphysema occurring merely from the strain upon the lung, and without any previous bronchitis or disease of the tissues; the strain being during the expiratory process, when the chest is expanded and glottis closed, so as to make a purchase for the muscles.

It will be seen that one of these theories supposes that the expansion of the air cells takes place during inspiration, and the other that it occurs during expiration. The first is also called compensatory or complementary, and is seen in cases when the lung has undergone contraction in parts as in chronic phthisis. The second is observable more frequently in cases of long-standing bronchitis. These theories do little more than account for expansion of portions of the lung, and both are dependent upòn a purely mechanical causation; but, after all, the most important form of emphysema is that when the whole of both lungs is affected, and where there is often no history of a previous bronchitis or cough. The case to which we allude is that where the chest has undergone a change in shape, where from the raising of the shoulders or the falling of the diaphragm it has become rounded or barrel shaped. In this case the lungs are of large size, are found after death filling the chest, feeling doughy to the touch, and when closely examined show the distended or ruptured air vesicles plainly visible to the naked eye. The whole lung may be found affected in this manner, and at the same time there is no appearance of contraction of any portion, nor need there be any history of bronchitis previous to the time at which the symptoms of emphysema first exhibited themselves. We are forced, therefore, to the conclusion that the cause of this universal structural change must have been inherent in the tissue of the lung itself. When examined it is found wasted and the blood-vessels diminished in number and size; the alveolar walls may be found to contain fatty granules, but this is scarcely sufficient to warrant the opinion that the pathological change is one due to fatty degeneration. When just now we said that this worst form of emphysema had not a purely mechanical origin, we would not deny that the inspiratory efforts when acting on a weakened tissue might directly produce the distension of the air vesicles. Why the pulmonary membrane should have become weakened is not always obvious; but in some cases we meet with fibroid changes in the lungs as if an interstitial pneumonic process had been present, and under these circumstances we can see how a stretching of the tissue might have been produced.

That a primary wasting of the tissue will cause the breaking down of the alveolar walls, and so give rise to emphysema is evident from its common occurrence in old age, when the whole lung is atrophied, and at the same time emphysematous. In such cases the whole thorax becomes diminished in size, and in this way are produced the stooping posture and sloping shoulders so characteristic of old age.

This senile change is often called *atrophous emphysema*, in order to distinguish it from the *hypertrophous emphysema*, where the chest is large and rounded. The term hypertrophy must be understood to imply no more than large.

The bronchial tubes are often found full of secretion; their walls thickened with great hypertrophy of the longitudinal fibres.

Hyperæmia and Congestion.—A congestion of the lung is constantly met with in cases where the blood is delayed in its passage; it is seen often in perfection in cases of paralysis of the chest where the spine is fractured, the lungs being exceedingly gorged, and sometimes to such an extent that the blood oozes through the bronchial membrane, and is extravasated into the tissue. In very many cases of bodies you examine you will find the posterior part of the lung much gorged with blood, and of a dark colour; this is due to the supine position of the patient some time before death and probably to the commencing softening of the tissues: this is known as *hypostasis*, and passes into an inflammatory state, presently to be mentioned.

A spotted condition of the lung, even if due to extravasation, does not necessarily show a mechanical impediment, for it may be due to a diseased condition of the blood or the tissues, and be of a *purpuric* character; thus, in true purpura, the lung as well as other parts of the body will be found spotted, both on the surface and the interior, also in many other organic diseases, where the same tendency to purpura exists, as well as in cases of manifest blood-poisoning, as pyæmia, &c.

A very long-continued congestion, as seen in heart disease, gives rise to a very peculiar condition, which is much in want of a good name to characterise it, for it is not mentioned by some authors, and by others classed with pneumonia. The name *splenization* having been first given to this condition, although since applied to a stage of inflammation, has generally been adopted here; you may remember that as a consequence of the long-continued congestion in heart disease, various necessary changes occur on the serous and mucous surfaces; and in the parenchymatous organs a remarkable hardening takes place, noticeable especially in the kidneys and spleen: in like manner the lungs are indurated, the organ feels bulky and heavy on removal, suggesting a hepatized tissue, but, on section and more careful examination, the structure is found dense and tough, and containing but little air, as you see in this wet specimen. When scraped it is fleshy, giving out no secretion, and instead of being granular the surface is glazed and shining. In parts of it, blood may be found actually extravasated, constituting apoplexy; but, without this, you may sometimes find a whole lobe solid, almost airless, and at the same time quite tough when an attempt is made to tear it. There are various degrees of this: when slight, the lung is red, and resembles a hardened spleen, and in this case a microscopic examination shows little else than stagnation of blood in some of the smaller vessels; subsequently the vessels become distended and tortuous, and thus encroach on the air-cells. In more extreme cases this redness is lost, and the lung assumes a browner shade; and when in this condition the microscope shows abundance of corpuscles and granule masses, a chronic inflammatory state is no doubt present. Of late years the name *brown induration* of the lung has been applied to this condition.

Sometimes in persons who have been suffering from disease of the brain, which interferes with the respiratory process, the lung may be found remarkably tough, fleshy, and firm. A remarkable instance of this occurred in a case of paralysis of the diaphragm.

Apoplexy.—If the pressure on the blood-vessels be very great, the blood is actually effused into the tissue, and we have what is called *apoplexy* of the lung—a very absurd name, but one now in general employment. The blood gradually oozes out, and infiltrates itself into the tissue, both into and between the cells, and there coagulates and hardens; so that it does not necessarily happen that any of the fluid escapes into the bronchial tubes, or that there is any hæmoptysis during life. Owing to this gradual exuding and coagulation of blood, the part thus affected becomes hardened, and the circumference is quite defined from the adjacent healthy tissue. The apoplectic condition is thus recognised on the surface of the lung before it is cut into; the part is enlarged, as it would be if hepatized, but instead of the induration passing off gradually into the adjacent tissue, it is defined by a distinct boundary, and thus it is felt like a tumour in the lung. A section at once shows its nature, and distinguishes it from hepatization; it is seen to be blood, and of a black colour, and resembles (to use the common simile) damson cheese, the texture of the lung being scarcely discernible, the branch of the pulmonary artery leading to the part being always plugged with fibrinous coagula. There are, however, cases where inflammatory products have been poured out, and thus a hepatized and apoplectic condition may be often combined; the section of an indurated lobe being white and granular in parts, and having small masses of blood in others; moreover, on the pleural surface it is not unusual to find a thin layer of lymph, showing the inflammatory process which has been present. Besides this, however, you may often find white fibrinous masses in the lung, indicating older effusions, the hæmatine having decomposed and become absorbed, leaving the more solid part of the blood as a white mass; and as this contracts, so does often the lung tissue with it; and thus is produced a puckered condition of its edges, with sufficient traces of the apoplectic state about it to show its pathology, In old cases of mitral disease these various changes in the apoplectic masses may be seen, but not often, the puckered state showing complete absorption, which probably is to be referred to some previous temporary attack of obstruction in the circulation. The favorite seat for apoplexy is the circumference of the lung, and especially the edges, as the lower edge of the upper lobe and upper edge of lower lobe; also the right lung appears to be more usually affected than the left; the explanation of which is not readily apparent.

Many of these well-known appearances of the lung in connection with heart disease have been shown of late years to have a different pathology; that these hardened and wedge-shaped masses are due to infarction or actual blocking of the pulmonary arteries by portions of fibrine carried from the heart; that just as infarction or embolic masses are formed in the spleen and kidney, in connection with endocarditis, so in like manner they may take place in the lung; that vegetations from the valves may be actually carried to a branch of the pulmonary artery, and there stick fast, or that smaller embolic particles washed off by the current of blood may give rise to coagulations in the arterioles, and so bring about the appearance known as an infarction. Probably the condition of lung above described, where the mass becomes developed and hardened, has had an origin of this kind. When in connection with endocarditis as of the mitral valve, infarctions are found in the spleen and kidney, which we call embolic, we cannot hesitate to give the same name to exactly similar appearances found in the lung.

It is remarkable that this apoplectic condition does not follow the congestion of bronchitis, but only that of heart disease; this is due probably to the seat of stagnation being different in the two cases: in the one, owing to the non-aëration of the venous blood, it can with difficulty pass through the capillaries; while in the other cases the blood passes through, but is detained in the capillaries and smaller pulmonary veins, whence it escapes into the tissue; the blood therefore, in apoplexy of the lungs, would be from the pulmonary veins rather than the arteries.

There is also another condition, which is sometimes called apoplexy, arising from a different cause, and producing a *spotted* condition of the lung, whereas that just described forms *circumscribed* masses. It arises from the entrance of blood into the air-passages, and its diffusion into the cells. The fluid passing down a tube, and entering one branch rather than another, small portions or lobules of the lung become infiltrated, producing this spotted appearance. It is met with in those who have died from hæmoptysis, and especially in those where the lung has been seriously injured by a fractured rib, leading to death by the drawing of blood into the tissue.

Œdema of the Lung.—This is a condition very frequently found in

Bright's disease, or where there is a disposition to dropsy; the lung is found filling the chest and heavy, and on a section being made a quantity of serum drains out, leaving the tissue healthy and firm. It is thus distinguished from the first stage of inflammation, in which the texture is very lacerable. Sometimes, however, when a person has been long dying, the texture may soften, and then if, as in renal disease, we know the lung is equally inclined to be œdematous or to be inflamed, it is difficult to say whether the serous infiltration be active or passive: this difficulty has given rise to the term *inflammatory œdema.*

Pneumonia.—This is generally divided into simple or *lobar* pneumonia, where the whole lung or a lobe is affected, and into *lobular* pneumonia, where distinct and scattered lobules are inflamed. We also speak of a *chronic* inflammation, where the connective tissue between the lobules is supposed to be primarily affected, leading to a general solidification and induration of the pulmonary tissue under the names of fibroid changes, cirrhosis, and analogous conditions. The lobular pneumonia is usually associated with a bronchitis, and is mostly met with in children, or under some particular circumstances as during measles. Of late years, and more especially since the microscope has been put in requisition, it has been maintained that this lobular or broncho-pneumonia is not a modification in form or degree of the ordinary pneumonia; but it differs from it in kind. It must be remembered that the mucous membranes undergo two kinds of inflammation, both of which may be observed on the air-passages,—the one where the secretion is corpuscular or muco-purulent, and the other where the secretion is fibrinous or croupous; as is seen in ordinary croup, where the larynx and trachea are covered with a membrane. In the one case the microscope shows a mass of cells formed by the proliferation of epithelium; whilst, in the other, the membrane is composed of fibrillated lymph containing cells in its meshes. Now, it is maintained that the inflammatory products poured out into the air-vesicles in ordinary pneumonia are of this latter character, and thus of late years many writers have given to it the name of *croupous pneumonia;* whilst it is also said that the lobular pneumonia partakes of the character of the bronchitis with which it is usually associated, that it is corpuscular in its formation, and would with more propriety be styled *catarrhal pneumonia.* It must be understood, then, that the term croupous pneumonia is applied by modern writers to ordinary pneumonia; whilst the term catarrhal is made to apply to what has hitherto been styled lobular pneumonia or more correctly broncho-pneumonia.

Simple or croupous pneumonia.—It was long taught that inflammation of the lungs consists in the exudation of a lymph into the interstitial substance or parenchyma of the lungs, but of late years the doctrine which was always taught by Addison in this school, who was the first to demonstrate the true nature of pneumonia, is now universally adopted. It is the air-vesicles or the alveoli constituting the dilated termination of the bronchi which contain the inflammatory products. Addison was led to his conclusion forty years ago by actual observations as to the seat of the exudation, by the structure of the pulmonary cells and by the proved absence of any supposed intercellular spaces into which lymph could be effused.

The changes which take place in the lungs are of the same kind as are witnessed in other structures, viz. first, an intense hyperæmia followed by a fluid and solid exudation. The change, therefore, which the organ undergoes is a most striking one, being none other than the conversion of a light spongy organ normally full of air into a heavy solid mass, and all within the space of a few hours. The different stages of the process are sufficiently well marked, and are traceable during life by various physical signs which accord exactly with the condition of the lung. They resemble what is seen in other structures during inflammation, viz. hyperæmia and vascular engorgement followed by an exudation.

The first marked change observable in the lung after death is that of *engorgement with serous exudation*, and this is usually called the first stage of pneumonia. On taking the organ out of the chest it is found to be more bulky and heavy than the healthy lung; it pits on pressure, and when cut through it is much redder from sanguineous engorgement, and a quantity of serum pours from it; owing to the lung still containing air the serum is frothy, and portions of tissue still float when cut off and thrown into water; if now the finger be forced into the lung it will be found to be readily lacerable, and this is a means of distinguishing it from simple œdema or dropsy of the lung. This softening is one of the best signs of inflammation, and it further shows that the fibrous tissue which forms the walls of the pulmonary air-vesicles is more or less involved in the inflammatory change, for although we give up the old theory of an interstitial inflammation, yet we must hold that in connection with the cell-formation which takes place the texture of the lung is loosened by an exudation into its meshes, in the same way as in inflammation of other structures; as, for example, of the peritoneum, where it may be observed that, although the exudation takes place from the surface of the serous membrane, yet the coats of the intestine are infiltrated with lymph, and are readily separable from one another. It is during this stage of serous exudation in the lung that fine crepitation is heard by the ear.

It is not usual until the lung has reached this stage in the inflammatory process that inflammation is recognisable during life, and it is rare for it to be seen earlier on the post-mortem table. Yet exceptionally this is the case, and it must be evident that prior to the exudation of any fluid there is a stage of simple engorgement. The air-vesicles would be encroached upon by the distended blood-vessels causing a harsh and deficient breath-sound. This early condition has been recognised by Stokes and confirmed by Addison, so that it would more justly be styled the first stage of pneumonia, and that to which this name is given would then constitute the second stage. We shall adhere, however, to the usual nomenclature.

The second stage of pneumonia, that of red hepatization.—As in the course of inflammatory effusions in other parts of the body the serous exudation at first poured out becomes more solid, so the same occurs in the lungs, the serum makes way for lymph, until the air-cells become completely blocked by it. The lung at this time when cut through no longer exudes serum, but a solid matter is squeezed from the cells; the blood-vessels, however, are still gorged with blood, which is in part stagnant within them; at the same time the secretion which has been poured out contains some blood-globules and hæmatine, and thus the organ still retains its red colour, though not so bright as in the healthy state. This dull red solid lung resembles, under these circumstances, a piece of liver, and thus the term hepatization has been given to it. On examining the cut surface it is seen to be slightly granular from the projection of the distended air-vesicles; it no longer emits any fluid or air on pressure, but rapidly sinks in water. It is also very lacerable when the finger is thrust into it.

The third stage, that of grey hepatization.—This is a further development of the previous stage by a more complete solidification. The air-vesicles are more tightly filled with inflammatory matter, the cut surface is still granular, sinking in water and easily lacerable; but the exudation contains less colouring matter, and the blood has been squeezed out of the capillaries, and thus the whole colour of the lung is paler. The grey colour, indeed, which the lung presents is due mainly to the natural pigment which it contains, and thus it happens that a hepatized lung is much darker in aged persons, where more natural pigment exists, than in children, where pigment is almost deficient; thus it is that the hepatized lung of children is white or yellowish-white. The nature of hepatized lung can be readily understood if you can suppose the minute bronchial twigs and air-cells tightly filled with albuminous material, and, indeed, it very much resembles the preparations in our museum where the organ is injected with tallow. When a lung is solidified or hepatized in the manner spoken of, it is seen filling the chest when the body is opened, as instead of collapsing as a healthy lung should do, it forms a mould of the thorax, and when removed the form of the ribs may be seen impressed upon it. Not uncommonly a slight pleurisy has accompanied the pneumonia, and then a delicate layer of lymph may be found on the surface of the lung and easily separable from it. A lesser amount of pleurisy is very commonly present where the membrane is seen to be opaque, whitish, or to have lost its lustre. This is so common that it is only when a considerable amount of lymph has exuded from the surface that the name pleuro-pneumonia is applicable. In comparing the solidified with the healthy lung it will be found that in some cases as much as four pounds of solid matter must have been rapidly exuded into the tissue. During life in this second stage of pneumonia there is an absence of all natural breath-sound, but in its place there is bronchial breathing and bronchophony with dulness on percussion.

We will now say a few words on the *histology* of the process which takes place in inflammation of the lungs. This, as might be surmised, has varied with the prevailing views regarding the exact changes which take place in inflammation wherever occurring. Before microscopic times pneumonia was regarded as an exudation of lymph into a supposed parenchyma of the lung, but when the organic nature of an inflammatory product was discovered the theory of Schwann was applied, and the ordinary explanation of a primary hyperæmia, followed by an exudation of serum called 'cytoblastema,' in which cells sprung up, was adopted. This theory of a spontaneous generation of cell-life continued until Virchow's dogma of "omnis cellula e cellulâ" took place, and the idea of proliferation of the endothelium of the alveoli of the lung was substituted for the older one; on this theory the hyperæmia must be regarded as secondary and necessary to the cell-growth. More recently the views of Cohnheim have been enforced by many pathologists, that inflammatory cells are none other than the white cells of the blood which have migrated through stigmata in the capillaries, and these views may be made applicable to the case of pneumonia. It does, however, appear to be a fact beyond contradiction that in pneumonia the endothelial cells grow and propagate, but this by no means is opposed to the later theory. If the air-vesicle of a case of so-called croupous pneumonia be examined it will be found to be filled with a firm and fibrillated substance in which are imbedded cells resembling the white cells of the blood, and others larger and of less regular shape which may well have been formed by the proliferation of endothelium; at the same time, owing to the engorgement of the vessels, some red corpuscles have escaped into the tissue, and some of them breaking up have stained the secretion red; this is observed in the rusty viscid sputum. At the later stages of pneumonia we may also observe the fibrous walls of the alveoli containing an exudation of cells, and these may in all likelihood have had their origin in the fibres themselves. It has long been a subject of controversy as to whether the bronchial or pulmonary arteries mainly share in the inflammatory process, and opinions are still divided on the point.

The stage of purulent infiltration.—According to the view which may be taken of this condition it may be regarded either as a sequel to the forms of inflammation just described or as a true fourth stage of pneumonia; indeed, if the hyperæmic state be regarded as a recognisable condition, the purulent infiltration would constitute a fifth stage. It is better, however, to be content with the three which are usually adopted. With the third stage, or that of grey hepatization, the inflammation has reached its height, and the attendant fever is departing; resolution takes place, the exuded lymph undergoes disintegration, the cells become granular and fatty, and thus the inflammatory products are removed by absorption or expectoration. Sometimes a simple absorption takes place, and the exuded matter may be taken up as rapidly as it was thrown out; this is certain from the frequent absence of expectoration and the return of the diseased lung to its healthy state. More frequently the secretion is thrown up, and the subsidence of the inflammation is shown by the change of the rusty viscid mucus to a thin and yellowish one. It is during this period, when the exudation is softening and passing into the tubes, that pneumonia is usually fatal, and it is frequently observed that the sputum instead of becoming lighter in colour has become purple or black. When a post-mortem examination is made and the lung said to be in a state of purulent infiltration, it is found not to have lost its solidity, for it will still sink in water, but when cut through, instead of being dry, it exudes a dirty-coloured fluid of a greenish-brown colour mixed with air. It is the hepatized lung softening and saturated with the fluid products; the exudation-cells are granular and broken up, and the alveolar walls full of the same material. Considering that in fatal cases of pneumonia this is the condition found, the stage of purulent infiltration has been regarded by some as a necessarily fatal one: that instead of a simple disintegration of the cellular exudation a further infiltration takes place into the alveolar walls, a loss of elasticity and actual softening follows with an impossibility of recovery. Yet there are those who with Hughes Bennett regard a change resembling this as a necessary termination of an inflammatory process, and therefore a purulent stage of pneumonia as an all but necessary one. Of course he would not imply, nor is it implied, that in the fatal form the term purulent is equivalent to the word suppurating; it signifies merely that the inflammatory cell or leucocyte becomes granular and fatty, and as some would further say, a pus-cell, but it does not mean that anything like purulent matter is seen in bulk or that abscesses are ever formed. Suppuration, in the ordinary sense of the word, does not take place as one of the stages of inflammation of the lungs. If an abscess is found in the lung the inflammation has not been of the ordinary or croupous variety, but it has been lobular and pyæmic.

It is a point of great interest to ascertain when the recoverable stage of pneumonia has passed and the fatal one begun. It may be true that the disintegration of the material is necessary, and thus theoretically a purulent stage must exist; but, as a matter of fact, mucous râles throughout the chest are regarded as unfavorable during the recovery of pneumonia, whilst the most favorable cases are those where absorption quietly takes place without any moist sounds. These are the reasons against thinking that the purulent infiltration usually found in fatal pneumonia is a stage which is reached in cases which recover; the probabilities are in favour of the hepatization in most cases not even progressing much beyond the red stage.

Lobular pneumonia.—This may occur in connection with obstruction of the pulmonary arterioles, as in pyæmia, and therefore is not a primary affection of the lung; it may also occur in connection with bronchitis, a form of malady mostly met with in children; and thirdly it may be seen as chronic affection laying the foundation for pneumonic phthisis.

Phlebitic and pyœmic pneumonia.—This is limited to individual lobules, or a group of them, their number being determined by the size of the pulmonary artery which is primarily involved, the peculiarity of this form of inflammation, being its rapid progress, and its termination in abscess, or sloughing. Its most frequent seat is the lower edge of the lobes, especially the inferior, where firm masses of consolidated lung may be seen, each mass averaging in size that of a walnut, but may be greater or less. On cutting through these masses the various stages of inflammation may be seen, one perhaps being in a state of red and another of grey hepatization, and another of abscess; thus these stages occur in rapid succession, and in the more favorable cases in the order already mentioned; and where recovery takes place, which is probably very rare, merely the hepatised state has been reached; but as a rule the disposition is to rapid disintegration, and thus within two or three days after the first onset of inflammation symptoms of sloughing or suppuration occur, the tendency being always to form an abscess. The disintegrating process probably comes on very soon after the first inflammatory stage; and no sooner is the exudation poured out than a rapid breaking down of the tissue ensues, and thus the lung may be found softened or sloughing with the surrounding parts only in the first stage of inflammation. It is worthy of remark how this form of disease occurs near the surface of the organ, as it does also in the liver, &c.; and the same is true also, though to a less extent, of adventitious products in general, the cause not being positively made out. Since this is the case, the pleura becomes involved in the inflammation, and thus every diseased portion of tissue is covered with a layer of lymph; and in some cases the whole lung is thus covered, or a general acute pleurisy has taken place. In this instance the abscess has generally burst into the pleura and so caused the serous inflammation. This form of disease, or lobular pneumonia, is generally connected with purulent absorption or infection, and is thus most frequently the immediate cause of death in pyæmia arising from injuries, operations, &c.; it was once thought that the pus was actually carried from the distant part to the central organ, and if no pus were present there, it was formed in the vein by phlebitis; but the disproof of this is, that all the stages of inflammation preceding the production of pus are present; there can be no doubt that something is carried to the lung from the infected part, and although this is not actual pus, it is probably some of the elements of pus or other infecting matter, for experiments have shown that the disease is produced after injecting such like material into the blood. When this has been done the immediate effect produced is a coagulation of the blood; or, if smaller quantities be used, it permeates into the smaller vessels, and there produces its effects, the first indication being a spot of congestion from the blood having coagulated in the minute pulmonary arteries; thus, on making a section through a lung the subject of lobular pneumonia, you will find, as we have already mentioned, the various results of inflammation; and preceding these, parts of the lung of a red colour; these patches of congestion being the first step in the process, and since in each mass the more rapid changes are towards the centre, an abscess may be found in the midst, the surrounding part hepatized, and the circumference of a red colour from this congestion of the tissue. It is highly important to notice these congestive patches, for we believe they always show blood disease, as already mentioned under fever and purpura; and in cases of purulent infection they precede the further inflammatory changes.*

On opening the pulmonary arteries the blood is found coagulated and the vessels plugged. In some cases it would seem that the fibrin had formed primarily in the larger trunks, and then being disintegrated had been distributed through the smaller branches, thus blocking them or setting up several centres of infection. In other cases it would seem that some deleterious matter circulating in the blood promotes its coagulation in the extreme vessels themselves. In whatever way the infarction occurs the fibrinous plug acts as an irritant, and sets up an inflammatory action in the lung tissue, at the same time, cutting off the blood supply, it favours a rapid sloughing, or even necrosis of that portion of the lung to which the blocked arteriole proceeds.

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* The importance of this condition was strikingly shown about two years ago, where it might have been made the means of convicting, or not, a man charged with murder. The prisoner was the captain of a ship, and charged with being the cause of the death of a boy who had sailed with him. The boy was brought to the hospital, and shortly died; he was covered with sores, arising from lashes he had received, but the immediate cause of death was a congestion of the lung, seen by a number of ecchymosed patches all over the organ, and associated with some bronchitis. There was very little scientific doubt that the state of lung indicated a blood-poison, and the only source of this was the abraded integument. As, however, it could not be positively said in a court of law that it did not arise from cold, the doubt went in favour of the prisoner.

Catarrhal or broncho-pneumonia.—This is a very important affection, and constitutes for the most part the inflammation of the lungs of children. Children rarely suffer from lobar pneumonia, but instead of this are affected by a bronchitis, which is apt to pass into the lobular and vesicular form. The inflammatory process extends down the tubes into the lungs, and at the termination of a certain number of bronchial twigs one or more lobules become affected, an exudation is thrown out into the cells, and the tissue becomes consolidated. If death has occurred at an early stage, and individual scattered lobules are alone invaded, the affection may be overlooked. If the lung, however, be carefully examined, numerous small portions of tissue may be found airless and of a redder colour than the surrounding parts; some of them may be merely collapsed lobules, but others will be found to be soft and granular, and exuding some lymph when squeezed, which again shows abundance of inflammatory corpuscles when examined by the microscope. At a later stage the inflamed portions become lighter in colour, and then may be seen on a section of the lung a number of small white bodies like millet-seeds; these are the lobules filled with exudation, and are likely to be called tubercles by the inexperienced eye; indeed, such vesicular pneumonia, when occurring with disorganization of the lung in phthisis, is likely to be called tubercle. When larger portions of the lung are affected a lobular pneumonia is sufficiently evident. The diffused character of the disease does not strike the eye so much as a uniformly consolidated lung, and thus its severity may be overlooked; but all the bronchial tubes should be opened out so as to examine the membrane or the secretion upon it; afterwards the tissue should be looked at, which by squeezing and close inspection may be found in parts airless, and in parts containing an inflammatory exudation.

Of late years microscopic examination is thought to have shown a different histological process between the two forms of pneumonia; in the one case corresponding to the croupous exudation in the air-passages, and in the other to the ordinary corpuscular secretion. So that in this lobular or broncho-pneumonia, of which we have been speaking, the air-cells, instead of being occupied by a fibrillated firm mass, are filled with corpuscles or leucocytes, which have had their origin in part from a proliferation of endothelium, in part from the migration of cells from the blood-vessels, and in part from secretion which has been sucked in from the bronchioles and plugged the alveoli of the lung-tissue.

A good example of the affection is met with in measles; its occurrence does not seem to depend upon the fact of the greater frequency of the exanthem in children, for it is met with in a most perfect form in adults.* Under exceptional circumstances there may also be met with in adults, forms of bronchitis which, spreading downwards, culminate as broncho-pneumonia; thus in tracheotomy and injuries to the air-passages this form of inflammation may be occasionally met with.

Chronic catarrhal pneumonia.—In connection with bronchitis in adults we occasionally find that after the disease has existed for some time a certain number of lobules may become affected, more especially at the base of the lung; the consolidated lobules form foci, from which the same process proceeds, until a large portion of lung has become hepatized. These portions being completely blocked up by secretion become hardened, and then undergo further changes. A section of the lung would be found to have scattered through it a certain number of yellowish-white masses of this hepatized tissue. In course of time, if resolution does not take place, these portions become still more dry, the blood supply is cut off, and they are converted into cheesy masses; these subsequently disintegrate or soften, and the lung tissues decaying with them disorganization of the lung ensues. This is *one form of phthisis* to which we shall presently refer. It is a question whether these caseous masses are the result of a chronic or slow albuminisation of the tissues or whether they result from a lobular pneumonia which was in the first place acute.

In all probability the *peripneumonia notha*, or bastard pneumonia, a disease about whose nature as described by the older writers there has been much controversy, was like this one of children a catarrhal or broncho-pneumonia, and thus we may notice in morbid anatomy, as well as in other relations, how senility is second childhood. The cases so called were those where a sudden and difficult breathing came on, which was speedily fatal, and evidently denoted an obstruction in the pulmonary tissues; and where the post-mortem inspection revealed only a bronchitis, or no evident consolidation of the lung. A careful examination of the tissue, in such cases, however, shows that the inflammation had crept down to the vesicular structure which terminates some of the bronchial twigs, and that these had been rapidly filled with exudation; at the same time other portions had collapsed from the bronchial obstruction, and then had ensued the fatal symptoms and rapid death.

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* In January and February, 1863, a Peruvian ship of war arrived in the Port of London, and soon afterwards measles broke out on board. About twenty of the crew were brought to the hospital, and three died. In these, most severe and universal bronchitis existed, the breathing was most difficult, and the expectorated matters were thick and purulent. The examination after death showed the same condition of the lung as is met with in children; that is, there were no extensive masses of consolidation, but there were small portions of hepatized tissue scattered everywhere throughout the lungs. There was acute bronchitis evidenced by the large amount of muco-purulent secretion and the reddened state of the mucous membrane.

Probably even less than this might be apparent where the case was that of a very old and feeble person occurring in the season of winter. For it may be observed both from the depressing effects of cold on the nervous system as well as from its directly injurious effects on the lung that the lung will collapse in large portions without any great evidence of bronchitis, and then assume the appearance already mentioned under the apneumatosis of childhood.

Typhoid Pneumonia.—It has been thought by some pathologists and writers on fever, especially the German, that in typhoid fever the whole body is peculiarly affected, and that various organs especially suffer. The ileum is the part where the diseased condition more particularly exhibits itself, but all other organs, we know, participate in a morbid state, and especially the lungs, recognised by the dyspnœa, cough, sanguineous expectoration during life, and the congested or inflamed condition found after death. The question is, is this affection of the lung peculiar—that is, is it due immediately to the typhoid process which shows itself in so characteristic a manner in the intestine—or is it merely due to the state of fever, and the consequent alteration of blood and tissues of the body? It is said, by those who believe the pneumonia to be peculiar, that the microscope shows characteristic cells of the same form as those in the ileum and mesenteric glands; but then, in objection to this, it may be said that, admitting the peculiarity of the intestinal affection, it is not satisfactorily shown how the deposit in it is peculiar, when viewed by the microscope, or that it is recognisable when examined. If, then, there be doubt about the microscopic peculiarities of the elements of the typhoid deposit in the intestine, and we trust rather to the naked-eye appearance for its recognition, how much more difficult is it to speak of the peculiarity of a similar affection of the lung, when the outward appearances are not so distinctive? It is thus thought by some, and with good reason, that the form of pneumonia found in typhoid fever is due merely and necessarily to the state of blood, tissues, and position of the patient. It is an appearance important to recognise, for though not, we believe, characteristic, yet, when associated with other conditions, assists in marking the disease. In all fatal cases of typhoid fever the lungs are found gorged with blood of a dark colour, and present a spotted appearance both externally and internally, as we have just mentioned is generally the case in blood diseases; it is a stage beyond this, however, which constitutes inflammation, but so intimately are the two connected that the one gives the other its peculiar characters, and, indeed, the general appearance of the organ and the part affected show how much the morbid state is due to a mere blood disease. Thus, as the congestion and ecchymosed appearance occupy the back part of the lung, so does the pneumonia, and it is, therefore, at the posterior part that we find consolidation; it is not hepatization of one lobe or another, as in ordinary pneumonia, but the posterior edge, and especially the part near the root of the lung, that is affected, and seldom passes beyond the stage of red hepatization; this is not uniform, but occurs in patches, and thus, if a section be made, it appears as if a number of lobules had been separately inflamed, leaving some healthy portions between them, or a certain number of lobules in a complete state of hepatization, while the tissue between is recently and acutely inflamed. If the inflammation extend beyond this, and affect a larger part of the organ, it may be looked upon as a complication, in the same way as an ulceration of the intestine following the typhoid process in the ileum. The reason, as we have said, why we are not sure that this condition is peculiar to typhoid, is that it may be sometimes met with in typhus; in the latter disease it is rare that the lungs are inflamed, but if so, in a very similar way. Also, as regards the enlargement and softening of the *bronchial glands* before noticed: this has been considered by some as a peculiarity of the affection; but certainly it is not so, for they are generally found affected in all cases of pneumonia, and more especially where a large part of both organs is involved.

Hypostatic Pneumonia.—This is a term used to express that condition of the lung which you so frequently see in the post-mortem room, affecting the posterior parts of the lungs. You will see, almost every day, lungs removed from the body which at their front parts are dry, spongy, and natural, but having the back parts doughy, heavy, and when cut exuding serum; this condition, conjoined with increased softness of texture, is that which characterises inflammation, and therefore the term pneumonia is adopted; as, however, it does not come on until the patient is dying of some other disorder, it is called *pneumonie des agonisants*, or *pneumonia morientium*, and must be looked upon rather as a result of the act of dying than a cause; and this is not difficult to understand when we consider the prone position of the patient, without any movements for days, and a disordered state of blood, as well as a commencing decay of the tissue, for you have already seen how, in fever, the back part of the lung, from a mere mechanical reason, is the part most liable to the morbid change; and then, if to these be added the decaying powers of the brain and nervous system, whereby the influence of the pneumogastric nerve is weakened, we gain some idea as to the cause of this process. In some cases we find the pneumonia has reached the stage of hepatization; and then a question arises whether this be the same form of disease further developed, owing to some accidental prolongation of the patient's life, or whether there be any especial exciting cause for the inflammation: if the pleura be affected, we should have no doubt in saying that such is the case. Thus, in Bright's disease, where an early condition of pleuro-pneumonia is so often found unexpectedly, there is a special cause for its production, and more than can be found in the mere act of dissolution.

Gangrene of the lung.—This may occur under various circumstances and to a variable extent. It is usually divided into *general* and *local*, or *circumscribed*, although these do not actually correspond to many cases frequently met with. The former is generally intended to apply to a general gangrene following inflammation, whereas the local arises from an accidental death of a particular part. In the majority of cases, however, which we see in the post-mortem room, it arises from the lung being involved in the disease of a neighbouring organ; thus, in cases of cancer of the œsophagus, you will generally find the adjacent parts of lungs, those near the root, in a state of hepatization or purulent infiltration, and, at the same time, sloughing or gangrenous; you will see, on section, a quantity of green, dirty fluid exude, the tissue broken up in shreds, and the odour indescribably fetid. This many of you know by handling such an organ, when you will find the odour attaching for many hours to the hands, and very different from the ordinary smell of decomposition. In cases of aneurism of descending aorta the same may occur, from pressure; and we have seen it, two or three times, result from caries of the spine. We might here warn you not necessarily to expect a gangrene of the lung because there has been a fetid expectoration during life, for this may occur, sometimes, in connection with bronchitis, which Dr Laycock states to be due to a compound of butyric acid which is formed in the lungs. In the *general form* of gangrene an ordinary idiopathic inflammation has preceded,—a pneumonia, in fact, has run on to gangrene; you know, in other parts of the body, this is one of the consequences, but it is so rare in pneumonia that we did not mention it among the usual terminations; it does, however, sometimes occur, and generally arises from some constitutional cause or epidemic influence; thus, we do not think we have met with a case for a considerable period, although a few years ago we had several such cases, but could not account for the occurrence except on the supposition of some epidemic as influenza; in these the pneumonia rapidly passed into gangrene. In these cases the dead part passes by insensible stages into the healthy, there being no distinct boundaries between them; the most affected parts being soft shreddy, and having a horrible odour, while the tissue around is soft and exudes a dirty-coloured fluid. In the *circumscribed form* of gangrene a portion of lung is found isolated from the surrounding parts, and is dead; this may result from a simple inflammatory process, and is due, no doubt, to the stoppage of the blood-vessel proceeding to it; this more frequently occurs in the chronic inflammatory processes in the lungs, and sometimes it arises, as Dr Addison used to teach in this room, from apoplexy of the lung, whereby blood being effused into the tissue its nutrition ceases, and death of the part results. You must see fresh specimens to recognise the true appearance, as these only show ragged tissue, but this drawing exhibits an isolated portion of dead lung, as in the wet preparation.

Chronic lobular pneumonia.—This is the disease already alluded to where, in connection with bronchitis, certain groups of lobules become consolidated, and are converted into yellowish-white masses; these are called caseous pneumonia and sometimes scrofulous. If they soften and cause disintegration of the lung they constitute one form of phthisis.

Local indurations of the lungs.—The nature of these will be further discussed in the next paragraph, but it may be here stated that in various forms of chronic disease of the lungs these indurations are met with; and in phthisical lungs the centres are surrounded by dense tissue. Those who are in the habit of making post-mortem examinations know how common it is to find consolidation of the apices which are at the same time adherent to the chest wall by thickened pleura. It would seem as if the connective tissue of the lung together with the walls of the air-cells had undergone an hypertrophy whereby the alveoli had become obliterated, and the true pulmonary tissue converted into a tough fibrous substance. On cutting through these indurated portions of lung they are dense and quite airless, and so exceedingly hard that it is impossible to thrust the finger into them. It may also be observed that wherever these indurations occur the tissue is darker than that of the surrounding healthy parts; this is due to hæmatine from the changes which take place in the blood during the inflammatory process. The microscope shows how the whole of the lung-tissue is united together in one mass.

Chronic interstitial pneumonia and cirrhosis of the lung.—There is no piece of pathological anatomy about whose true nature so much controversy has been aroused as that of chronic affections of the lungs; one main question being whether the different appearances met with, often in the same lung, are essentially of one kind, or whether they are due to two or more causes, since different observers may see in the same lung tubercle, scrofulous deposits, caseous changes of inflammatory products, true interstitial pneumonia, and cirrhotic processes. It is therefore necessary to speak separately of the conditions known under these names.

As distinct from the more ordinary pneumonic changes seen by a cell production in the alveoli, and even from the more chronic forms of a similar character, there is a condition tending to induration of the tissue where a production of fibrin or fibro-plastic material swallows up and envelopes the whole respiratory portions of lung. The term interstitial is scarcely correct, as it might imply a process limited to changes in the interlobular connective tissue, whereas the process of which we speak commences in the alveoli and their walls. A portion of lung is seen to have entirely lost its spongy character, and becomes hard, dense, and fibrous; also, owing to the presence of pigment, it was styled by Addison iron-grey induration, and he also likened it to Aberdeen granite. The microscope shows a fibro-nucleated material occupying nearly the whole of the pulmonary structures, although within the alveoli there may also be found cells and nuclei. It is a question whether the new product has its origin in the fibrillation of a cell formation, or whether it does not spring up entirely in the walls of the alveoli and blood-vessels, and then slowly proceed after the manner of a growth. This indurated fibrous tissue sometimes puts on a granular form, and thus it has often been described as associated with tubercle, and, further, it has been maintained that the grey tubercle of fibro-cellular formation has a pathological and histological origin perfectly similar to that of this new tissue.

It is not uncommon to find lungs indurated in this manner throughout a large part of their volume, and to this the name chronic pneumonia is usually applied. In some cases, as above mentioned, the surface is granular, and to this the name tubercular is given; in other cases distinct cheesy masses are interspersed with the fibroid material, showing that a corpuscular inflammation has once been present in the air-vesicles. Sometimes the tissue ulcerates and cavities are formed, and to this condition the term chronic pneumonic phthisis would be appropriately applied. If there is no unequivocal tubercle present the term fibroid phthisis is sometimes employed. It is a disease met with in adult age rather than in youth, and frequently associated with similar fibroid changes in other organs. Occasionally, however, the same condition may be met with in youth, as, for instance, in that of a lad whose lungs are preserved, and which were streaked, as it were, with adventitious fibrous tissue.

An extreme form of the indurated lung which we have been describing is known by the name of *cirrhosis*. Many pathologists have been unwilling to separate it from the former, but have been generally forced to do so from the fact of the disease being so much more complete and from often being confined to one lung and unaccompanied by any changes in other organs, showing it to have a constitutional origin. The chronic pneumonic, or fibroid disease is never confined to one lung; it is, therefore, called phthisis, and has a constitutional origin, whereas the cirrhotic disease has often a mere accidental cause for its origin. In a well-marked cirrhotic lung the organ is shrunk into a small compass with all or nearly all the respiratory spongy tissue destroyed, all being swallowed up in a dense fibrous tissue. This is so dense that it cuts like fibro-cartilage, showing a surface smooth and grey, which is generally intimately blended with an enormously thickened pleura which also cuts like cartilage. In the majority of cases the section shows the lung permeated with thickened bronchial tubes, many of which are also immensely dilated so as to form large cavities. There may be distinct excavations in the tissue also. The main peculiarity of the disease is the fact of the conversion of the spongy lung into a dense mass of fibrous structure with enormously dilated bronchial tubes running through it. There has been much controversy as to the relation between the dilatation of the tubes and the induration of the tissue. Laennec believed, and he has still many followers, that in cases of enlarged tubes with a dense intervening tissue the cause was primarily in the bronchi, which being inflamed, a softening of their walls occurred, a giving way of their boundaries, with a necessary compression of the intervening tissue. Corrigan subsequently gave his opinion that an inflammation and contraction of the pulmonary tissue constituted the primary change; that in consequence of the contraction of the tissue the tubes became expanded as a compensatory result. In favour of the view that the cirrhotic process is one of the tissue and altogether local is shown by the fact, which in some cases seems undoubted, that it has begun as a pleurisy; the pleura has slowly become thickened, and at the same time the fibrous thickening has continued inwards through the whole lung until it has become universally indurated. Sections of lung may show the fibrous element surrounding the bronchial tubes and the blood-vessels, and the walls of the air-cells thickened until the whole structure of the lung is involved in the new fibro-nucleated tissue. It seems to have been a true interstitial pneumonia without the occurrence of any more active form of inflammation.

Other forms of chronic pneumonia.—It will be presently seen under the head of phthisis how opinions vary as to the nature of many of the chronic formations found in that disease. There are those who would regard them as varieties of chronic pneumonia, and would, therefore, rightly describe them in this place. It will be also observed that under the heading, acute pneumonia, a lobar and lobular pneumonia have already been mentioned, and that the latter may be of the chronic form: and also that there is a chronic pneumonia whose nature is believed to be of a totally different kind, being, indeed, the result of a fibroid and interstitial change; but it remains for further discussion to ascertain whether there is not a chronic pneumonia whose nature partakes more of the ordinary lobar, or so-called croupous kind.

We refer now to cases where, after an illness of a few weeks, during which time all the evidences of consolidation are present, the patient dies, and the lung is found (to use Addison's expression) uniformly albuminised. The section is not soft, lacerable and granular, as in the acute grey hepatisation, but it is smooth, solid, and tough. The air-cells are full of fibrillated lymph, and the exudation cells may have commenced to be granular and fatty. It is a condition of lung very liable to break down, and thus constitute a case of rapid phthisis, and on the other hand it is a condition from which recovery may take place, and the lung be completely restored. That there is a chronic pneumonia of such a kind can scarcely be denied when it is remembered for how long a time all the signs of consolidation may endure, and then a complete restoration take place. We must, therefore, believe that there is a true chronic pneumonia whose origin is an ordinary inflammation and exudation into the alveoli, and whose appearance is best denoted by the term *uniform albuminization*. Such chronic pneumonia, although occupying a large part of the lung, may be essentially of the chronic lobular kind, in which the various foci of inflammation have run together.

Phthisis.—Although we wish to do little more than give a description of the morbid anatomy of the phthisical lung, yet we are necessarily compelled to make some allusions to the different opinions which prevail as to the character of the pathological process which leads to the destruction of the organ, since the signification of the terms in use can scarcely otherwise be understood.

The term phthisis is generally used to imply all chronically destructive diseases of the lungs, and is equivalent to the popular expression consumption; the word chronic being applicable to cases of rapid consumption as well as to those whose progress is counted by years. It might well, therefore, be supposed that the appearances differ considerably according to the duration of the disease, the constitution of the patient, his age, and other circumstances.

In a large majority of cases it may be observed that the destructive processes in the lung appear to be due to the disintegration of a softish yellow-white material which has been formed in the air-cells. This has choked the tissue, cut off the blood supply, and then passed through the stages of softening. It has been styled by some yellow tubercular or scrofulous matter, implying by this term a deep-seated constitutional cause for its occurrence; whilst by others it has been regarded simply as a product of inflammation which has choked up the cells of the lung and then undergone a fatty degeneration styled caseation; the inflammation, it being stated, differing from ordinary inflammation in being vesicular or lobular, and also of a low type from occurring in feeble or scrofulous constitutions, and sometimes even being the result of a lobular inflammation arising from pyæmic infection.

Associated with the production of this material and a destructive process, we recognise a tendency to reparation, or at all events we generally meet with a large amount of new fibrous tissue which circumscribes the cavities, hardens the lung and binds together its lobes, at the same time fixing the whole organ to the walls of the chest. These well-recognised products of inflammation suggest a further reason to some for believing that the material already spoken of, which is thrown out in the phthisical lung, is of an inflammatory nature.

With these two elements in the phthisical lung, the inflammatory and the questionably inflammatory, there is generally associated a third element known as the miliary tubercle, appearing in the form of a hard, grey translucent body growing in the walls of the air vesicles.

As regards the microscopic appearances of these morbid products, an examination of the grey translucent hard tubercle just spoken of, shows it to have been formed in the walls of the alveoli; it is composed of a number of cells contained in a fibrous matrix or reticulum. The general resemblance of this to a lymphatic gland structure, has caused the name adenoid or lymphoid to be given to these tubercles by some pathologists, and even more than this, since it has been said that the smaller bronchi are rich in lymphatics, and that these tubercles grow at their extremities; that the tubercles actually take their origin in the lymphatics themselves. The cells composing the miliary tubercles are small, and about the size of lymph corpuscles; but besides these there are sometimes found larger cells, styled giant-cells which some have thought to be characteristic of the formation.

The softer material first spoken of, which is called yellow tubercle, is seen filling the air-cells. It has been formed within the alveoli and termination of the bronchi, from the endothelium lining these parts, and is composed of a mass of irregularly shaped cells consisting of epithelium and lymph corpuscles. These have become opaque and fatty, and block up the air vesicles.

The products found in the phthisical lung, which all regard as inflammatory, may be of various kinds; sometimes resembling what is seen in the hepatization of recent pneumonia; sometimes resembling the firmer albuminous material of a slower inflammation, and sometimes the denser fibrous tissue which conglomerates all the structures of the lung.

The earlier observers, as Laennec and his followers, finding the miliary tubercles not only in the lungs in connection with a phthisical and destructive process, but also as simple deposits in other organs, believed that these little bodies constituted the basis whence sprung all the other morbid changes which take place in the lung. Tubercle suggested to them a deep-seated cause in the constitution of the patient and often of an hereditary kind. In finding also that this tubercle might soften and change in colour, they advanced the doctrine that in phthisis these small bodies were first formed in the lung, that they then underwent a kind of ripening process, becoming soft and yellow, and so change into the yellow tubercle which, all are agreed, is the most important element in phthisis. Any other material which was found of an undoubted inflammatory character, was considered to be secondary and due to the irritation caused by the tubercles. Such was the pathology of phthisis long taught in the schools.

Objections soon rose to this doctrine and more especially on the part of Addison, but these objections were not very widely propagated until the recent writings of Niemeyer became known. Addison maintained that it was possible to trace in the various morbid deposits in the lung all stages between an ordinary inflammatory product, and the so-called soft tubercle; that the latter therefore was only one form of the result of inflammation. Phthisis, in fact, was the result of a pneumonic action. He taught that in persons of a weakly constitution, and more especially in that known as the tuberculous or scrofulous, an insidious inflammatory process might take place by which certain lobules or vesicles would become filled with an inflammatory product, that resolution not taking place as in ordinary pneumonia a fatty metamorphosis or caseation would ensue, that this would be followed by softening, owing to its deficient vitality or want of blood supply, and finally a general breaking up of the tissue and destruction of the lung. It was this degenerated material seen in the lobules or vesicles which was by others called tubercle. It might be true that the inflammation was not always of the ordinary kind, that a constitutional cause, whether inherited or not, existed to account for the slow and scattered form of the inflammatory deposit, and it might be true that the latter was unhealthy or cacoplastic compared with the euplastic lymph of ordinary lobar pneumonia. As regards miliary tubercle, Addison said they were sometimes present and sometimes not; they had no direct histological relation with the softer material which he called inflammatory, and others yellow tubercle; but their presence merely showed the proneness of those who had them to consumption. Such persons were apt to have tubercles form in the parenchyma of the lung, and these same persons were liable to a low form of pneumonia which not terminating in resolution, but in destruction of the lung, made them the subjects of phthisis. But this evidence of the predisposition to consumption in the presence of tubercles was by no means necessary. Phthisis constantly occurred in persons having the peculiar constitution in which tubercles were found, but also equally in other persons of broken-down constitutions, or where the system had been debilitated by such causes as diabetes or alcoholism.

On this view all the varieties of phthisis can be explained, from acute pneumonic phthisis to that form where the lung becomes completely indurated owing to the organisation of inflammatory products; and more than this, since there is evidently some relation between all these varieties and miliary tubercle, the latter must also be regarded as arising under the influence of inflammation.

Of late years Niemeyer has more fully developed this view, and has maintained that the destructive process in the lung is due to the formation of a material which is essentially inflammatory; having its origin in a lobular pneumonia, a scattered inflammation of the lung found in connection with bronchitis, and therefore, a result of broncho-pneumonia, or as he styles it, a catarrhal pneumonia. He believes that the material is formed by a proliferation of the endothelium of the alveoli of the lung, undergoes a caseous change, softens and disintegrates. The miliary tubercle he regards in the light of an accidental growth which may be present or not. The tubercle may precede the formation of the inflammatory material, but very often accompanies or follows the phthisical process. Niemeyer may be regarded as the strongest advocate for the inflammatory origin of phthisis.

At the present time opinions are divided amongst these theories of the phthisical process. It would be admitted by all that an inflammation is present in most cases of the disease, and more especially in the chronic forms where indurations are found. But the question in dispute has reference to the connection between the true miliary tubercle and the softer material known as yellow tubercle, which constitutes the very essence of phthisis. There are those who, with Addison, see between the so-called miliary and yellow tubercle very little immediate kinship; they find, for example, both lungs and other organs of the body stuffed with miliary tubercle, but without any tendency for the tissues of these organs to disorganise; the more tubercle, indeed, which is present the less is there of the condition which can be called phthisical. The essential product of phthisis and tubercle, they therefore say, must have a different origin. There are, on the other hand, many pathologists, whose names carry weight, who maintain that these two products are essentially alike; they are both intimately connected with the phthisical process in the lung, they form slowly after the manner of growths, there is a specificity about them, and, therefore, they must be separated from simple inflammatory products.

It is true, they say, that there may be some difference in the composition of the grey tubercle and the softer yellowish deposit; the one may be more of a lymphoid character, and composed of cells contained in a fibrous stroma, whilst the other may be more purely cellular; the one be of slower growth and more clearly arising in the walls of the alveoli, whilst the other shows a greater rapidity of growth, arising more especially in the endothelium of the air vesicles, but nevertheless they maintain that the material formed under these latter circumstances has a closer relation to the true miliary tubercle than to inflammatory product. An inflammatory product is a mere endothelial proliferation in the alveoli, whilst the true phthisical product, of whatever kind, involves the alveolar walls, and then pursues a certain course. It has a specificity, a nature, and a history of its own; it is, in fact, more allied to a growth and thus deserves the name of tubercle. Even if it be true that much of the caseous matter is of an inflammatory origin, yet in the midst of all there is the true tuberculous element which grows and destroys.

It will be seen, then, that there are those who not denying that inflammatory products are found in the phthisical lung, yet maintain that the yellowish-white material, admitted by all to be the real destructive element, is truly tuberculous matter, as well as that which is usually recognised by the name; and there are those who would limit the term tubercle to the latter or grey semi-transparent body, asserting that the other product is the result of a low form of inflammation or consists of a cacoplastic lymph.*

If we take a large number of phthisical lungs and regard them in the rough, we shall perceive how all these products of which we have been speaking are intimately associated, and how it has been easily suggested to the observer to look upon them as but different forms and stages of one simple pathological process. We may see, for example, a lung considerably disorganised the subject of acute inflammation; another in which there are albuminous deposits not distinguishable from what are seen in the different stages of hepatization; and another in which there are undoubted inflammatory products associated with the before-named yellowish deposit whose origin is disputable, and with these perhaps also miliary tubercle. In all these varieties we may see also fibrous thickening, and indurations of a chronic kind.

In a typical case of phthisis in a young person which has existed from one to two years, the lung as a rule has been first affected at the apex, and the disease has gradually progressed downwards, usually in one lung, in advance of the other. Consequently the greatest disorganisation is found at the upper part whilst the deposit which has constituted the origin of the disease is seen in its recent formation in the lower lobe. Whilst destruction has been going on reparative changes have also been in progress and, consequently, these are best seen where the disorganisation is greatest, and thus considerable induration of the tissue may be seen at the upper part of the lung, where also the pleura, covering the apex, may be extremely dense while the lower lobe is adherent by soft adhesions. In the upper lobe, involving the extreme apex, there may be a large cavity, its walls dense and adherent to the chest; these walls being composed of thickened pleura and the remains of indurated pulmonary tissue; the lower part also of the cavity next to the lung may be surrounded by a similar hard material. Below this there may be other and smaller cavities of more recent formation, and in consequence the tissue which circumscribes them is not so dense.

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* In the controversies which have taken place as to the tubercular or inflammatory nature of phthisis, there can be no doubt that with many the former view supposes a constitutional disease, and the latter a purely local or accidental one. Addison insisted as much upon this great clinical distinction as he did upon the purely anatomical difference of what he considered to be the two forms of the disease. He saw as one form the case of a young person who, in a predisposed constitution, fell an early victim to consumption of the kind where the disease comes on insidiously and progresses from apex downwards, and he saw as another form of phthisis the case of a middle-aged person who, from causes undermining his constitution, such as diabetes, would be liable to a pneumonia, exhibiting itself as a consolidation of a lobe of the lung, and afterwards disintegrating. He might also see the second or inflammatory form not necessarily end in speedy disintegration, but the product, instead of undergoing caseation, fibrillate, producing an induration of the lung. It can scarcely be denied that, clinically speaking, two such classes of cases exist, and therefore those who see in every disorganising lung a material which they distinguish from inflammation, and call tuberculous on account of its peculiar growth and destructive tendencies, would deprive the word tubercle of much of its original meaning, and make these two forms of disease essentially alike. The controversy would strengthen the opinion that all nomenclature of disease should be clinical rather than pathological.

As regards the cavities in the lungs, you will find these vary according to the age of the disease; in the more recent deposit, a mere hollow will be found containing pus and broken-up material; while in the upper lobe, where the disease is older, the cavity may have a hard wall and have travelled down the organ to some extent. During its formation the bronchi and bloodvessels are differently affected; the latter become closed and forming hard cords, are seen traversing the vomica from side to side, 'and thus hæmorrhage is precluded. If you cut through one of these you will see the obstructed vessel within, and thickened externally by indurated lymph, and sometimes remains of lung tissue upon it. Whilst the vessels are being obstructed, a different process occurs in the bronchial tubes; these are destroyed, together with the lung tissue, and are so gradually worn down even with the walls of the chamber, and are found opening on its surface, and through these the contained matters are discharged. Sometimes a cavity is round and circumscribed, with hardened walls; at others it proceeds downwards, forming a long straggling space with the obliterated bloodvessels traversing it. As we before said, in the indurated parts of the lungs, the cavities there found communicating with the tubes may have resulted in an expansion of these tubes, although when large it is difficult to prove this; for all trace of the original bronchial mucous membrane is gone, and the cavity could not have been produced without a corresponding wasting of the parenchyma around it, and thus it is often useless to discuss the manner by which such cavities are formed. It is only, however, in indurated parts of the lungs that such can occur, and, therefore, in cases of chronic pneumonic phthisis the cavities found within it are generally expanded bronchial tubes, the indurated tissue being little prone to soften.

The lower portions of the lung may be occupied by the so-called scrofulous deposit or cacoplastic lymph, and some of this may be already softening and small cavities becoming formed. Portions of lung may be occupied also by a morbid product, not in the form of distinct particles but as a tranlucent gelatinous material which has been regarded by some as the matrix of true tubercle.

Having given the above as a typical case, we may mention that a great variety of appearances may be met with, dependent mainly upon the acuteness or chronicity of the disease. Thus sometimes cases of phthisis are met with where the disease has run a very rapid progress; when the destructive process has gone on without any compensating preservative action. There has been no induration of any part by the development of connective tissue, but the whole lung is crowded by soft yellow albuminous or scrofulous matter which has undergone so rapid a softening that the whole lung resembles a sponge soaked in purulent matter. There is no tubercle properly so called, and the only morbid product is the whitish-yellow material which is undergoing caseation. The cavities if formed are ragged and soft, their sides being composed of the disintegrating lung. Towards the bases there may be considerable masses of the morbid material not yet softened and which, according to Addison, are not distinguishable from the ordinary grey hepatisation of the lung.

We meet also with cases even more acute than this and where the illness has extended over a few weeks only, and has been attended throughout by febrile symptoms. Such cases have been called acute pneumonic phthisis. The only physical signs being those of a pleuro-pneumonia, where consolidation is discernible and often in the lower lobe. Subsequently, a breaking down occurs accompanied by the ordinary symptoms of phthisis. Addison described several cases in connection with his essays and drawings of phthisis; for example, a young man aged 21, had been ill two months from the time of getting cold; he had high febrile symptoms and all the physical signs of disorganising lungs. The lower lobes of his lungs were found solidified and covered with lymph; the section showed them occupied by deposits of a low organisable character, pale yellowish, in small circumscribed masses, and breaking down. In two other cases of young girls, with a similar history, the consolidation occupied large portions of the lung tissue, and each albuminous mass was softening in the centre. Such cases Dr Addison believed to hold an intermediate place between true tubercular phthisis and pneumonia. The more general or lobar the consolidation, the more rapid is the inflammatory process and the nearer is its approach to ordinary simple sthenic pneumonia; the more it approaches to lobular or vesicular consolidation, the more chronic is the process and the greater resemblance it has to tubercular or scrofulous disease.

In many forms of cachexia and debilitating disease this form of phthisis is met with; thus Addison's typical case of pneumonic phthisis came from a patient who had suffered from diabetes. The lung had been uniformly albuminised and then broken up into cavities. In these cases of acute pneumonic phthisis, the inflammatory process may still be seen in progress in parts of the lung in the form of small distinct granular masses of grey or red hepatisation.

In contradistinction to the last-named form we have chronic pneumonic phthisis, a disease which may have lasted for some years and mostly met with in the later years of life. In these cases the lung may contain cavities and circumscribed yellowish masses of albuminous material scattered here and there which would be called inflammatory or tuberculous respectively by the advocates of the two theories of phthisis; but the lung for the most part is indurated, the tissue has become conglomerated into a hard mass by the formation of a connective tissue which binds all the structures together; and which also is of a dark colour from pigmentation. Such a lung is very hard and requires considerable force to be used in order to cut it through. The albuminous circumscribed masses just spoken of may be found also to have undergone a cretaceous change or become mineralised.

There is also a variety of this chronic phthisis in which portions of lung are not only indurated but granular, or as if composed of a mass of tubercle. Sometimes a rounded mass the size of an orange will be met with, dark in colour, very dense in the centre and apparently composed of tubercles bound together by fibrous tissues radiating from a point. Away from this the tubercles become less in number and the tissue less dense until at the circumference both these elements altogether cease. Much controversy has taken place respecting the nature of these masses. Some maintaining that with the development of the true tubercle, the fibre constituting its matrix grows in excess, and thus the tubercles become welded together by it. Addison maintained that these masses represented the remains of a pneumonia in which induration, puckering, and granulation had taken place and thus the appearance of tubercle was merely assumed. In many of these cases the lung tissue is found much puckered and contracted and the adjacent pleura much thickened.

Modifications and lesser amounts of these morbid conditions may be constantly found in those who have died of various disorders. Thus it is by no means uncommon to discover the apices of the lungs indurated by fibrous tissue or occupied by some circumscribed masses of albuminous matter; and the apex at the same time firmly adherent to the chest walls. There may also be found the round masses of conglomerated tubercles.

In the two last-named varieties it will be seen that the lung tissue is much indurated by the infiltration into its substance of a fibrous material which converts it into a solid mass. This is regarded as the result of a prior inflammatory process. But there are cases in which both lungs are found infiltrated and streaked with fibrous tissue and in which there has been no evidence of any inflammatory process. Such cases have given rise to the belief that they constitute a peculiar form of phthisis. That in the more ordinary case the patient is young and the product which is formed in the lung is of cell structure and prone to decay, whilst in the above named, the patient is older, of a different constitution, and liable to the occurrence of fibroid changes in various tissue. So there is gradually produced, and *ab initio*, a fibroid tissue which in time overwhelms all the healthy structure of the lung. After a considerable time an ulcerative process may take place, resulting in the formation of cavities. Opinions are at present divided whether this fibrous induration of the lung is simply the relic of a long past inflammation or whether it be not a special pathological condition arising under special conditions. We have already referred to this under chronic pneumonia and cirrhosis.

Again, we may find one lung entirely destroyed in its proper structure by the production of a fibrous tissue, and rendering what was once the lung so changed and hardened that the name cirrhosis has been given to it. The whole lung has become, in fact, one hard mass of fibrous tissue closely adherent to the chest by thickened pleura of the densest structure. In some cases it is evident that the morbid change has commenced in the pleura on the surface of the lung, and the latter has become secondarily invaded. The disease thus appearing to be due in many instances to a local cause has tended to remove it from the class of cases which we have been hitherto considering, and which all arise under hereditary or acquired constitutional conditions. This also we have already described.

You will constantly find remnants of disease in the lungs of those we examine, showing that the affection under consideration is curable; what we generally meet with indicating this is the apex of a lung adherent, and its surface puckered, and, on making a section, the tissue is indurated, dark, and only in part permeable to air, and often containing earthy or chalky matter. The probabilities are, that in these cases a deposit of tuberculous matter, with some inflammatory exudation, occurs at the apex, and, instead of this softening or increasing, it ceases under some advantageous circumstances, and dries up, but the indurated tissue remains, and with it any earthy constituent of the tuberculous matter. It has been thought by some that this puckered tissue, having often a cicatriform appearance, represents a closed cavity, but there is no proof that this ever occurs. Probably, if a cavity has once formed, it never closes. The earthy matter found in the lung is sometimes expectorated during life, as found in these specimens. Curiously enough, a kind of mould may form in old phthisical cavities, as described by Bennett and Bristowe; this parasitic fungus (but probably there is more than one) is mentioned by Küchenmeister as the aspergillus pulmonum hominis.

It may be observed that although fatal hæmorrhage is more liable to occur at an advanced stage of phthisis, yet severe hæmoptysis is much more frequent in the early stages of the disease. It then arises from ulceration of some of the smaller pulmonary vessels or from some local congestion of the bronchial arteries. At a later period when cavities are formed the blood-vessels have become sealed, so that a fatal hæmorrhage occurs under other circumstances. It might arise from the vessels on the walls of a cavity, but not unfrequently from a large branch of the pulmonary artery which has become aneurismal. A small sac, the size perhaps of a bean, may have formed on the hardened walls of an old vomica and then ruptured.

Syphilitic Disease of the Lungs.—That syphilis will attack the lungs there can be no doubt, but exactly what share it takes in many of the destructive or phthisical diseases is still a question. Distinct syphilitic gummata may sometimes be met with in the substance of the lungs, having the same formation and character as similar deposits in the liver and testes and other favourite seats; the coexistence of these in different organs can leave no doubt as to their nature. Again, associated with these deposits and fibroid thickening and indurations we meet with similar chronic changes in the lungs; these are less characteristic, and hitherto have been included under the general name phthisical. A very remarkable case occurred under Mr Birkett of a woman who had a large syphilitic sore on the thorax, which involved the ribs and penetrated the chest. It was found that the same fibrous substance which formed the floor of the ulcer involved the pleura beneath and the lung itself, so that the latter contained the same fibre-tissue as the syphilitic sore. Such a case proved that an interstitial or chronic pneumonia may have a syphilitic origin. In any given case of phthisis occurring in a syphilitic person a question may arise as to the true character of the morbid change in the lung which has led to its destruction, and at the present time tests are wanting to enable us to assert that products, usually called fibroid or tuberculous, have been produced under the influence of syphilis. That such an influence has been exerted is proved by clinical rather than pathological facts, where patients come before us presenting all the usual characters of incipient phthisis or rather pneumonic consolidations, which are speedily and remarkably cured by specific remedies as iodide of potassium. Under these circumstances there can be no doubt that exudations occur peculiar in their nature and formed under a syphilitic influence, for on no other conjecture can it be explained that they would be so readily removed under special treatment. Whether the morbid products, if they could be then seen, would present any special appearances capable of recognition is a matter still open for inquiry. If death occurs at a later period from disorganisation of the lungs with all the ordinary symptoms of phthisis, then our ordinary means of investigation are put into requisition to discover in what way the morbid appearances differ from those which are usually met with; whether the fibroid changes are peculiar, and whether softer deposits usually styled tuberculous should here receive a specific name.

At the present time we must admit that there are true syphilitic gummata to be met with in the lungs; also that there are more chronic formations removable by remedies, which are probably of a fibro-plastic character, and which if not removed develop into true fibroid changes and produce the phthisical lung; thirdly, there may be a more general and diffused hepatization of the lung due to the syphilitic influence, and this is a form occasionally met with in children.

The *hæmoptoic* variety of phthisis is one which has quite recently been added to the nomenclature, but not yet been accepted by all pathologists. It is a disease said to have its origin in an accidental coagulation of blood in the tissue of the lung. Hitherto the opinion of Laennec has been almost universally adopted, that hæmoptysis is a symptom of incipient tubercular disease, and even when no physical signs are apparent the disease has been assumed, since in so many cases a disorganisation of the lung has sooner or later followed. The more recent opinion is that an accidental hæmorrhage into the lung may itself lay the foundation for phthisis; that depositions of blood may set up a chronic form of pneumonia, and this undergoing the caseous degeneration may lead to the disorganization of the lung; or even that the blood-clots themselves by their presence and obliteration of the vessels may undergo disintegration, and so give the starting-point for farther changes. The subject of phthisis ab hæmoptoë is still under discussion, and requires much more illustration than it has yet received.

Under the term phthisis are also included cases of disorganisation of the lungs induced by the entrance into the lung of irritant substances contained in the air, and thus we speak of *miners' phthisis*, *millstone grinders' phthisis*, or what appears to be the same thing, grinders' asthma. The term phthisis is used because a disorganisation of the pulmonary tissue is apt to occur, but from the nature of the morbid process this class of disease might with equal propriety have been described under the name of chronic pneumonia. The subject has been fully investigated by Drs Peacock and Greenhow, and they have found that particles of foreign matter, mostly mineral, are drawn in with the air into the tissue of the lung; it there lodges, sets up a chronic inflammation accompanied by the usual exudations, and that these softening and disorganising destroy the lung after the manner of phthisis or a bronchial phthisis.

In the coal miners' phthisis the lungs are black, a black juice can be squeezed out of them, the secretion from the tubes contains pigment in the form of granules or in cells, and pigment may also be found in the alveoli and their walls. A section of the lung shows pigment and inflammatory products surrounding the tubes, as well as siliceous matter and alumina. In a case of potters' asthma or phthisis Dr Greenhow found the same minerals in the lungs as were contained in the dry dust given off in the process of china scouring, and in a case of French millstone grinders' phthisis Dr Peacock found siliceous and carboniferous particles, some of these being sharp and angular corresponding to what was discovered in the dust of the workshop. In the Cornish miners' lung Dr Peacock has been unable to find any foreign matters, and thus he regarded the disease to which they seemed liable as ordinary tubercular phthisis.

Pigment, or Spurious Melanosis.—As a rule, the colouring matter in the lung increases in proportion to age, and thus a child's lung is comparatively white, while an old person's is of a blackish hue; consequently, all diseases at the two respective ages show a difference in colour; this is especially the case in chronic inflammation of the organ, where the older the person the darker will be the colour of the disease, the pigment having its origin in the hæmatine thrown out during the process of inflammation. The most remarkable condition, however, connected with pigment is that called spurious melanosis, or, vulgarly, black spit, occurring in those who have breathed a carbonaceous atmosphere. The fact of a black lung being found in those who had breathed a black atmosphere, at once led to the conclusion of a connection between them; but then the difficulty arose, how came it to pass? In the first place, let us inquire as to the nature of the pigment. In an ordinary lung you will find it forming dark patches in the tissue; and you know that the secretion from the bronchial tubes contains pigment. You observe in the sputum some black specks, and if you place this under the microscope, you will have a very striking object: you will see the ordinary mucous globules, with epithelium, &c., and amongst these some larger cells, various sizes, as you see in this drawing, containing numerous black particles, showing that in the secretion the colouring matter is, so to speak, organised—that is, it is contained in organic cells. In persons who work in coal-mines, and at trades where a carbonaceous atmosphere is respired, the expectoration is inordinately black; and in some cases these persons fall into a bad state of health, and die with disorganization of the lungs, called in general terms, phthisis, which may be of the tubercular or chronic pneumonic character. Apart, however, from such disease and disorganisation, the lungs in such persons may become blackened, as you see in these specimens from miners of

Newcastle; in these no change can be discovered beyond the whole tissue being quite black, as if the organ had been soaked in a black fluid. In a case which came before us not long ago, of a man who had worked for many years in a gas factory, the lungs were indurated and very black, so that a quantity of dark juice came out when the part was scraped, and when placed on paper looked like Indian ink. In the same way the bronchial glands were enlarged, and converted into black masses, which could be squeezed into a paste composed almost entirely of pigment. In this lung there was considerably more than in an ordinary case of chronic pneumonia, since it stained the finger when touched. The pigmentary matter has been analysed by Christison, and found to consist of a hydro-carbon, and to be combustible like coal; and even said, on microscopic examination, to resemble coal dust; but the great difficulty is in ascertaining the direct connection between the carbon in the atmosphere and that in the lung. This difficulty in seeing how it can penetrate the tissue has caused some to think that the production of pigment was merely a secondary or accidental circumstance, seeing that it is a natural constituent of the lung, and is always produced wherever a chronic inflammation exists; and thus that the irritating quality of vicious atmosphere breathed by the miners is merely instrumental in setting up this disease. Such explanation, however, is not satisfactory, for it is especially in those who breathe a black air that this is found; and moreover, as we have just now shown you, a lung may be blackened without being diseased. Does the impurity, then, contained in the air actually pass through the air-passages into the lung tissue, and even to the bronchial glands? Such a supposition appears less improbable since the experiments made, to show that inorganic and insoluble substances like carbon, sulphur, or chalk, can pass through membranes, and may be taken up from the intestinal canal, and enter the lymphatics and mesenteric glands. Animals placed in a smoky atmosphere soon have their lungs blackened, so we must believe that the particles penetrate the lung to get to the interlobular tissue and go on to the lymphatics. It is thought that the black matter breathed by the miners is due to an impalpable form of carbon given off by their lamps, and not to the dust of coal; and in corroboration of this it may be mentioned, that at the present time there is a man in the hospital who is suffering from bronchitis, with one side of the chest dull, denoting probably chronic pneumonia, and the expectoration is very black; now, this man has worked for years in the vaults of the London Docks, where torches and candles are continually blazing; and this is not the only instance of the kind which has come under our notice.

Morbid Growths.—These, for the most part, are secondary, and are of all kinds; probably there is no description of tumour occurring on the surface of the body but what may recur in the lungs, and thus we meet with cartilage, bone, myeloid, &c., in these organs, as well as the more malignant growths; but very few of them occur as primary diseases of the lungs.

Cancer.—Primary cancer of the lung is a very rare disease, but, when the organ with the neighbouring tissues has become involved in carcinoma to a great extent, the general term of intra-thoracic cancer has been given. You may be surprised, perhaps, to hear the opinion that primary cancer is uncommon, considering how frequently we meet with carcinoma of this organ; but the explanation is this, that in those cases where this constitutes the sole or principal disease, the morbid product has commenced in the bronchial glands or peribronchial tissue, and involved the lung secondarily. We would, therefore, say that cancer of the lung is generally of *two kinds*,—that in which the parenchyma is filled with cancerous masses, *secondary* to a similar affection in another part; and that in which the lung is occupied by a mass of disease, perhaps limited to the chest, and which, in contradistinction to the other, may be called *primary*, although it has its origin really in the bronchial tissues. The first form, where the cancer is found diffused through the lungs, is met with in cases where the disease has originated elsewhere, as after the removal of a cancer from a limb, or following the same disease of stomach, liver, or other abdominal organs; also, often after cancer of breast, either as distinct and separate deposits, or by actual contact from growth through the thoracic parietes. The cancer may be scattered as minute tubercles throughout the tissue, or in the form of large nodules, and very frequently seen as projecting flattened nodules on the surface of the lung. Sometimes, as before mentioned, they appear on the pleura, as hard, smooth plates, resembling cartilage.

The second form of cancer, or *primary*, as it is generally called, because, perhaps, the only part of the body affected, or from constituting the disease from which the patient suffers and dies, has its origin, in most cases, in the bronchial glands, or in the cellular tissue around them, and then creeps into the parenchyma along the outside and course of the tubes; generally, a large mass may be found encircling the root of the lung and involving the organ at that part; if a section be made, the cancer will be found forming a layer around the tube, and proceeding with it, sometimes, to the most distant part of the lung; thus, if the organ be cut through, no disease may be found in the parenchyma, but the sections of the tubes will be seen encircled by a dense mass of white tissue. In some places, particularly if the disease be of the very hard or scirrhous kind, the tubes will be found contracted, and in some cases cancerous tubera will be discovered growing through the tubes into the interior. In some cases, where you find a large cancerous tumour in the chest, and it would appear most unlikely that the disease proceeded from the root of the organ, you may, on removing the parts, find the lung compressed, and comparatively unaffected, while the adventitious growth has sprung up on one side, and really in the way named. In these specimens you see cancer on the surface of lung. A *true primary* cancer of the lung is so rare that we have only one specimen to show. Here will be seen a lung with small nodules scattered throughout it, and none others observable in the body.

In most cases of cancer of the lung the pulmonary tissue is also the subject of a destructive pneumonia. Portions of lung are hepatized and breaking down. This may arise from the irritation of the new growth, from plugging of the bronchial tubes or from implication of the pulmonary nervous plexus.

It is not unusual also to find chronic pneumonic deposits, and then apparently a cancerous is associated with a phthisical disease. The question of their concurrence thus arises, but there is little doubt that tubercle and cancer may occur together in the same lung. We have seen a lung containing large phthisical cavities also the subject of cancer.

The form of cancer mostly met with is *medullary carcinoma ;* vascular, giving out much milky juice, it is seldom, however, very soft, unless it grows quite out from the surface; when the disease exists to a less amount, it is often harder, contains a fibrous matrix, and may be styled *scirrhous ;* an example of this you saw the other day, in a woman who died of cancer of the breast. *Melanosis*, we have repeatedly before said, may be nothing but cancer accidentally coloured by pigment, as we think is the case in this example, where it returned in the lungs after removal of the eye. We have one specimen of colloid disease of the lung which had its origin in the bronchial glands.

Epithelioma.—In cases of epithelial cancer of œsophagus, we have seen, in one or two instances, the adjacent part of lung involved in the disease; and in one case, distinct deposits at a little distance. In one case where it was found in connection with the same disease in the larynx we had reason to believe that the germs had been sucked into the pulmonary tissue and there propagated.

Recurrent or malignant fibroid — sarcoma. — These are generally secondary to the same disease elsewhere, as when, for example, a recurrent fibroid tumour has been repeatedly removed from an extremity, the disease at last appears in the lungs. It is not very common, and may be called semi-malignant. In this specimen, you see such tumours, which proved fatal in the lungs after similar ones had been removed from the skin several times; they consist of hard, round, firm fibrous masses. This specimen shows a primary disease of this character, where the lung is involved in a fibrous or fibro-cellular growth; it is the only case of the kind we have seen.

Osteosarcoma.—This disease is merely a fibroid tumour, with the addition of bone, owing to its proximity to osseous structure; sometimes, after removal, the same form of growth appears in the lungs, as round tumours composed of fibre and bone, the latter being true osseous structure, and constituting, generally, their circumference, as you see in this lung, and several other specimens.

Osteoid cancer, malignant osteoid, or osteoid-chondroma.—This is a bony growth of a malignant nature, as we have already described, and it may occur secondarily in the lungs, after removal from another part.

Bone may, however, occur as a primary disease, although so rarely that it may be a question whether or not a malignant osseous tumour has not been present as a primary disease in some part of the body, and overlooked. We have already alluded to a case where bony plates were found on the surface of the lung.

Cartilage, or enchondroma.—This is not common in the lung, either as primary or secondary disease, but is occasionally met with under both forms; thus, the remarkable specimen in St Bartholomew's Hospital, related by Mr Paget, where the lungs are full of cartilage, was secondary to enchondroma of the testes, whilst the specimen of cartilage before us removed from the lung purports to be a primary and local growth, and here is another where the disease began in the bronchial tubes and then penetrated the lungs as in cancer.

Myeloid.—In this specimen you will see a myeloid disease of the lung. This form of growth is probably much less malignant than any we have mentioned, and this constitutes the first specimen we have seen, the disease being generally quite local and innocent. You will see by the mode of growth how it differs from cancer: instead of being in the substance and infiltrating it, it springs from the surface, and thus you notice the pendulous character of these tumours; there is no other specimen of growth resembling it. They show the true myeloid structure, consisting of a red-coloured soft material.

Various combinations of these may occur in the lungs, as in the original tumours, and thus in this specimen there are masses composed of cancer, myeloid, and bone, as in the primary growth which was removed from the leg.

Tubercle.—This we have already mentioned, and a specimen you see here. Its favourite seat is the upper part of the lung. Much has been written about the seat of tubercle, and its frequency of occurrence in one organ rather than another. Louis has said, that after the age of fifteen it can never occur in any organ without the lung being affected. He is probably right, and might even have carried the age still lower, or speaking in general terms, and allowing only a very few exceptions, tubercle may be said to be never or very seldom developed in the interior of the body without the lungs being affected. We have already said, that in acute hydrocephalus (and the same is true in most other tubercular affections) the lungs may nearly always be found to contain tubercles. The age mentioned by Louis is interesting, as being that of puberty: the time, certainly, in which tubercle is wont to show itself in these organs: indeed, we are convinced of the truth of the observation made several years ago by Dr Barlow, that tubercle is developed in a particular organ according to its activity, and thus it is that the brain of infants is the most susceptible; at a later period the abdomen; and at the age of puberty, when the chest expands, the lungs are the most susceptible; and thus phthisis is comparatively rare before this period. Tubercles are very rarely congenital; only a few cases have been recorded. In cases of *acute tuberculosis*, where the whole of both lungs is stuffed with tubercles from apex to base, death occurs long before any disorganizing process can ensue, and the affection is generally accompanied by a bronchitis, and the lung found red and very vascular.

Hydatids.—These may grow in the lungs as well as in other organs, but in the majority of cases where they are expectorated, the seat of the parasite is the liver; it having made its way upward through the diaphragm into the lung. Sometimes death ensues, but very often the lung and liver coalesce, and recovery takes place. In this specimen you see a cyst making its way through from the liver, and in this other the hydatids are apparently formed in the lung.

DISEASES OF THE ALIMENTARY CANAL

TONGUE

Malformation.—In extreme malformation of the oral cavity in unviable abortions the tongue may be absent or only represented by a stump at the back of the cavity. The tongue may be congenitally unnaturally fixed by adhesion to the floor of the mouth, or its frænum may extend too far towards the tip or be too short.

Hypertrophy.—The tongue also is subject to a very curious hypertrophy—*Macroglossia*—which is often congenital, but may increase after birth and give rise to great suffering, requiring partial removal, and, perhaps, causing death by inanition or suffocation. It generally affects especially the anterior part of the organ. The tongue is found greatly swollen, it hangs out of and stretches the mouth. Virchow first showed the nature of the disease: the enlargement is caused chiefly by dilatation of the lymphatic spaces in the organ, so that it becomes like a sponge, with channels full of a clear fluid. The muscle is generally not increased, but there may be irritative increase of the connective tissue and thickening of the epithelium. The whole condition is like that of the legs in elephantiasis. In a few cases the tongue has been similarly enlarged by greatly-developed *nævus.*

Inflammation.—You may meet with general inflammation, or *glossitis*, as a part of a diffused inflammation of the mouth, through injuries, or salivation, or smallpox, &c.; but sometimes you find such glossitis as an isolated affection. An example of this you may see here, in an immense enlargement of the tongue, which was the cause of a woman's death in the course of a day or two. We have found pyæmic abscesses in the tongue. In more chronic cases, an effusion occurs gradually into the substance of the tongue and its swelling may remain for a considerable time. *Ulceration* of a simple character also occurs, or the ulceration may be aphthous, tuberculous, or syphilitic. Tuberculous ulcers may rarely extend from the larynx to the root of the tongue; *syphilitic* ulcers may be either in the form of condyloma or superficial ulceration, or else they may be in the form of deep excavations, with hard walls, very like cancer. This condition it is most important to recognise. Tongues have been "successfully" removed when iodide of potassium would have cured them. Such cancer-like syphilitic ulcers do not begin on the surface, but arise in the substance of the tongue, and soften down, breaking through to the surface, as subcutaneous gummata will soften and break through to the surface of the skin.

Morbid Growths.—We would impress upon you that every deep-seated knot, however large, in the tongue should be assumed to be syphilitic, though *hydatids* do occur in the tongue, as well as a very few other deep-seated tumours.

Cancer is rather frequent; it is by far most commonly *epithelial* cancer; it mostly does not spread beyond the contiguous parts and adjacent lymphatic glands. This form of disease affords a very striking object for the microscope, in the aspect of the large single or mother cells amongst the muscular fibrillæ. You meet this kind of cancer here in various forms, from hardish warts, arising in the papillary layer, to soft tumours, arising in the submucous tissue, and erroneously regarded as encephaloid cancer. Curious congenital *pendulous* tumours are sometimes met with on the tongue; they have a structure like that of nasal polypi.

MOUTH AND FAUCES

Malformation.—The mouth rather frequently shows malformations. We shall not ask your special attention to its conditions in unviable fœtuses, in which it may be continuous with the nasal fossæ and even with the orbits, or the lower jaw may be absent. There are many faults of its structure compatible with life. Thus, the mouth may be made small or closed by cohesion of the lips, or these may be ill-formed and imperfectly distinguished, the orifice being stiff and small on that account; or a fissure may extend the mouth on one or both sides nearly to the ears. But the most common and important malformation is that known as *hare-lip*. In this condition a fissure, or two symmetrical fissures, are found in the upper lip corresponding in position with the junction of the fœtal intermaxillary with the superior maxillary bone. The fissures may extend here into the jaw through non-union of these bones, and if the case be bad the portions included between the fissures may be absent, or much reduced in size, causing a great gap in the upper lip. The *uvula* may be *bifid*, as in this specimen; or the *soft palate* may be "*cleft*," through non-union of its halves, or the cleft may implicate the *hard palate*, when it may rarely be seen on both sides of the vomer, or else be single; this condition may complicate hare-lip. The soft palate may be absent.

Inflammation.—We will first speak of the affections of the back part of the throat, diseases in which the pharynx is also commonly involved, for these parts are intimately connected in their pathology. One of the commonest diseases is simple sore throat; if this be severe it receives the name of *cynanche tonsillaris*, inasmuch as the violence of the inflammation is spent on the tonsils, so that they swell greatly through œdema and increase of their lymph-gland-like structure; if yet more severe, the inflammation often ends in suppuration, producing tonsillar abscess or *quinsy*. A sore throat may arise, also, from other causes, as *scarlatina;* in this form, great swelling results, which does not terminate in abscess, but rather in sloughing if the case be grave, although a superficial suppuration or ulceration not infrequently happens. The structure of the scarlatinous tonsil is not microscopically to be distinguished from that of the swollen Peyer's patches of enterica (Pl. VI). In ordinary cases the inflammation has a tendency to affect the Eustachian tube and internal ear. A slight sore throat occurs, also, frequently in the exanthemata, fevers, erysipelas, &c. We have seen suppurating tonsils and glands at the root of the tongue in rheumatic fever. Virchow describes a great enlargement of the tonsils and glands at the root of the tongue in hydrophobia; this he calls *Lyssic angina.*

After repeated attacks of cynanche, the tonsils become permanently *enlarged*, constituting a very common and troublesome affection. You may see this in the youngest infants, so that it appears, sometimes, to be congenital; in older persons, these glands are readily excised, and numerous specimens you see in this bottle; they almost constitute tumours; when large they are chiefly composed of lymphoid structure like that natural to the tonsillar follicles, but sometimes they consist of tough fibre tissue, developed in the substance of the gland. Their surface presents this peculiar cribriform appearance from enlargement of the mouths of the crypts around which the follicles of the glands are situated. Occasionally, chalky concretions form in the tonsils, as you see here; they are composed of phosphate of lime. Sometimes, also, the whole soft palate becomes thickened and indurated. Sometimes the tonsils are replaced by the scars of old ulcers.

Suppuration may be found not only in the tonsil, but at the back of the pharynx, in a *phlegmonous* form; it may then have a deep-seated cause, as disease of the bone; for caries of the cervical vertebræ very frequently causes a retro-pharyngeal abscess, as we have already mentioned. But in young children retro-pharyngeal abscess may occur as a primary affection, or as a consequence of scarlatina, or some other fever. The abscess forms a swelling at the back of the pharynx, pressing forwards on the glottis so as to endanger suffocation. We have seen great abscesses arise here from the irritation of ill-placed wisdom teeth, also in states of exhaustion without such local cause.

Diphtheria.—This is an affection described by Bretonneau many years ago, but it has only lately been recognised in England as a distinct form of disease, although it probably corresponds to the malignant sore throat of former times. The local peculiarity of the disease is the formation of a membrane, hence the name given to it; and, in consequence, the term diphtheritic has been, of late years, used very generally for any inflammation attended by a membranous exudation like that of croup, whether seen in the throat, intestine, eye, or in other parts. In various diseases accompanied by sore throat, a considerable extent of pellicular exudation may occur; but diphtheria appears to be altogether peculiar: in it a membranous pellicle is formed, which may be of any degree of consistence, from an almost creamy softness to that of the petals or even leaves of plants; when pretty firm it sometimes can be removed in one piece, and then represents a perfect cast of the part of the mouth or throat, leaving the surface beneath livid and congested, or, perhaps, ulcerated; actual sloughing is rare. The disease begins, first of all, by a redness on the throat, and soon white patches are seen here and there, until they coalesce into one membrane, covering the pharynx and posterior nares, and extending into the trachea and bronchi. But topical varieties are met with frequently, wherein the disease fastens primarily on only one of these parts and extends but little to the rest. The corresponding lymphatic glands are enlarged considerably. Some uncertainty prevails as to the character of the pellicle, some being of opinion that it is a mere exudation, such as is often seen on an inflamed mucous surface, while others consider that it is a vegetable fungus. Opinions seem in this country almost decided that the part played by the fungus is small. Trousseau ignores it altogether, while Letzerich has more recently described the fungus under the name Zygodesmus as the determining cause of the disease, which, he says, in its first stage shows germination of the spores, in the second stage a formation of a white pellicle, partly of fibrin, partly of altered epithelium, and partly of the glistening filaments of the fungus, while in the third state the affected parts break away gradually or by sloughing; with this view he recommends treatment by rubbing alum in with the finger covered with linen. It is very certain that in some forms of throat affection, attended by a white pellicle, such a fungous parasite is often found, as we have several times witnessed; but, then, it is more than doubtful whether these instances have exemplified true diphtheria. For, indeed, it is often very difficult to distinguish anatomically between diphtheria and ordinary croup, or severe aphthous stomatitis. As to croup, some attempt to found a decisive distinction on the limitation of the false membrane, which they say in croup does not extend to the pharynx. But if this is so, then many cases of the sporadic form of tracheal inflammation must be regarded as diphtheria, although they are not contagious nor attended with enlargement of the glands, nor with constitutional prostration, nor with any other of those terrible peculiarities that make the distinction of diphtheria from croup of practical value. We have often found in cases of sporadic croup the false membrane forming patches on the soft palate and tonsils. Another distinction which is mentioned is the greater adhesion to the mucous surface which the diphtheritic exudation shows, while in croup the false membrane can be very easily separated. But we have found that in cases of undoubted epidemic diphtheria, when the pellicular disease descends the trachea, the false membrane is easily separated, while in croup cases, when the membrane extends to the fauces, it becomes closely adherent, so that the degree of adhesion appears to depend on the character of the mucous surface affected, adhesion being greater on the tough squamous epithelial surfaces than on the softer ciliated epithelium of the trachea. The chief distinctions of tracheal diphtheria from croup are in the precedence and low character of the constitutional fever, the greater implication of the lymphatic glands, and the contagious nature of the disease; no sufficient purely anatomical criteria are yet known. Aphthous pellicular stomatitis (p. 360) is always easily distinguished by its history and circumstances.

Specific ulceration.—The various forms of sore throats may go on to ulceration: cynanche often leading to small simple ulcers in the tonsils, &c. You may have observed amongst the out-patients, lately, some very severe cases of ulceration, which could be scarcely called by any other name than phagedænic; one that of a boy, where the whole soft palate was destroyed, as if by cancer; he was highly scrofulous, but whether such could be called *tuberculous ulcer* we could scarcely say.* As a rule, tuberculous ulcers of the throat proceed from the larynx, and thus are unlike the syphilitic, which may attack the pharynx or larynx independently. In the former, you find the disease has commenced in the larynx in connection with phthisis, and then has spread over the glottis into the pharynx and throat. The *syphilitic ulcer* is much more common. We will just mention the occasional occurrence of primary chancre in the mouth, but syphilitic ulceration is generally of two kinds:—First, you meet with ulcers during the eruption or so-called "secondary" stage; these are generally callous, well defined, rather excavated ulcers with a greyish floor, they are commonly symmetrical and may appear in any part of the mouth, though they generally prefer the tonsils. The second kind are in the "tertiary" stage, perhaps, long after the syphilitic fever is over. These are unsymmetrical ulcers and may spread widely and deeply, so that in cicatrising they may lead to serious stricture, or they may destroy life by their extent. We met a case where the left half of the hyoid bone stuck out of such an ulcer. These may affect the larynx low down, or destroy the soft palate and creep upwards into the posterior nares, or involve also the roof of the mouth. This form of ulcer is attended with gummatous thickening of the subjacent texture, sometimes it perforates and leads to fistula outside.

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* A similar case has lately been in the hospital, where the ordinary tonic treatment was of no avail; but a speedy cure took place under mercurial inunction, which was considered (with other facts) sufficient evidence of the ulcer being a consequence of *congenital syphilis.*

Stomatitis vesicularis, or aphthæ.—This is a well-known affection, occurring in children, and in adults towards the close of many diseases. A white pellicle appears, at first in small patches, and extends until it covers the tongue, mouth, and fauces. If the pellicle be examined, it will be often found to be composed of the mycelium and sporules of a parasitic fungus called *oïdium albicans.* It is not, however, quite clear how far such a parasite is a mere accident of the disease, or an essential part, but transplantation of the fungus has produced thrush in healthy children, so that there is no doubt it plays an important part in causing the disease, though a weakly state of the child and an acid state of the oral secretions are also important elements in its production; hence attention must not be fixed on the local condition only. Indeed, there is evidence that the fungus is not always present in these pellicular diseases, being absent in some very bad cases of throat affection. Aphthæ may extend into the pharynx and œsophagus. You must remember that an analogous form of growth is found between the teeth of those who do not habitually use the toothbrush, but who are in perfect health. To this last parasite the name *leptothrix buccalis* has been given by Robin.

Stomatitis follicularis is generally of little importance; it appears in one or several swollen glands that may form ulcers, round and well defined; in the fauces they may be attended with a few days' feverishness in delicate children, and create an unnecessary alarm of diphtheria. We have once known such follicular ulceration extending throughout the cheeks and lips as a very obstinate disease in an adult.

Stomatitis ulcerativa.—This is a very common form of disease, and one which you see constantly among the out-patients, where ulcers may be found in various parts of the mouth, but especially along the gums. It is connected with a constitutional disturbance, and you will easily recognise the disease of which we speak if we tell you it is that which is so readily cured by the chlorate of potash.

Pustular inflammation.—You find, as a rule, in severe cases of smallpox, that the throat is covered with pustules.

You meet sometimes with *acute œdema* of the pharynx in Bright's disease. This is very dangerous from its tendency to involve the larynx.

Stomatitis gangrenosa, cancrum oris, or noma.—This disease is one you occasionally see in the hospital; it affects children, most frequently from three to five years of age; it is generally fatal. It occurs mostly after some fever, as measles, typhoid, &c., making its appearance as a slight swelling and pale red patch on the cheek; this rapidly becomes deep red, then decays and turns black. The disease, however, has meantime made a greater progress on the mucous surface, which exhibits a slough while yet the skin is red. The sloughing goes on rapidly within the mouth, so that the whole inside of the cheek becomes a foul grey and most fetid pulp, the opposite gums become invaded, and the teeth loosen at last. The cheek breaks down, and the tongue and pharynx are exposed, while the teeth fall out. In typical cases the disease commences thus in the substance of the cheek, and its mucous surface is first affected. But sometimes gangrene starts from the gums. Trousseau describes gangrene as arising from these and reaching the cheeks after "diphtheria" has been several months in the gums. Yet this disease is quite different from ulcerative stomatitis. It is attended by severe constitutional disturbances and perhaps delirium, but not by much suffering. It may be associated with similar disease of the genitalia. Recovery rarely occurs; but if so, it is generally with exfoliation of parts of the jaw. We have examined several cases of the disease after death, but have failed to find any good explanation of the affection, either locally or constitutionally. The blood-vessels of the face proceeding to the part have been carefully dissected, but nothing in them has been discovered to suggest a reason why even in great constitutional debility this part of the body should be especially affected; and as regards any general cause, the same difficulty exists, since sometimes no organic disease can be found, although in two or three cases a fatty liver has been met with. If this were always so, we could scarcely tell what amount of importance should be attached to the fact, until we first knew in what time this change in the liver may occur; also to what amount the fat accumulates before any functional alteration accrues, and a condition is arrived at which can be called morbid.

Morbid Growths.—You sometimes meet with hypertrophy of the submucous glands forming *adenoma*, these may reach a large size, and cysts may form in connection with them. Such adenoma has occurred also on the tongue congenitally. Here are several specimens of *warty* growths, or papillomata, from the soft palate. They are more frequent on the lips. This drawing of one shows loops of blood-vessels covered with scaly epithelium; indeed, they resemble papillar or warty growths elsewhere. Sometimes a growth on the palate is flatter, harder, and more fibrous, so as to be called a *condyloma*, as you see in this specimen.

Polypi are mostly found growing in the pharynx and upper part of the œsophagus. These have the same structure as nasal polypi.

Epulis.—This is an ancient name for tumours on the gums; but tumours on the gums are of many kinds, and have a general likeness to tumours in other parts; hence a difficulty in settling which tumours of the gums are to be called epulis. There is no constant practice on this point, as you may judge when we tell you that Virchow, we think rightly, gives the term a topographical significance, and calls all tumours of the gums "epulis," so that there is myxomatous epulis and sarcomatous and fibromatous epulis, while the College of Physicians in its nomenclature goes curiously to the other extreme, and after placing all possible tumours of the gums under No. 385 makes epulis No. 386, but gives it no import, so that it stands like a venerable title without estates.

Using the term, like Virchow does, as an equivalent for tumours of the gums (which really come from the periosteum of the jaw) you will find that many of these are of the nature of *sarcoma*, but especially of that variety in which Lebert, while framing his description of fibroplastic tumours, first figured the cells which were called *myeloid*, but are now known as *giant cells.* In such kinds of epulis the cells may reach a size so great that they are visible to the naked eye. These cells are now known not to be peculiar to any kind of tumour; nevertheless, the great size and clear definition of them in these tumours of the jaw give such tumours a special interest. But an epulis may be simply a fibroma, or may be compounded of fibroma and sarcoma; it may be observed to recur very often after removal. It is very firm, springs from the periosteum, and is covered by gum tissue; and this specimen has a few plates of bone in it, a condition not unfrequently met with. Here is another specimen which came from a man who was said to have epulis, but the tissue is *epithelial cancer* growing from the gum; it rapidly returned after removal. It is an old question whether such cancer should be called epulis. This is a question of words, yet epithelial cancer is so well recognised, and its tendencies so well known, that it is always advisable to give the name when it is seen to be applicable.

This form of disease, *epithelioma*, you know, is very common on the lip, and its structure is seen then to great perfection.

Carcinoma occasionally attacks the soft palate and tonsils, as in a woman lately in Mary ward. We have met with *lymphomatous* cancer of the tonsils along with the same disease in the spleen, &c.

We have already shown you a *horny* growth from the lip. *Angioma* or nævus is frequent there. We need not, indeed, recount to you all the diseases of the skin which the skin of the lips suffers, like other parts of the tegument. *Lupus* may extend within the lips and reach the tongue.

We might here allude also to another morbid state, which you will do well to look for sometimes on the dead subject as well as the living; we mean the *lead line* on the gums; this you recognise as a blue or dark slate-coloured rim along their edges, as seen in this drawing. If examined more closely with a lens, you perceive the colouring matter has a dotted appearance, from the papillæ being first affected.

Ranula.—This name is given, much in the same loose worn-out way as that of epulis on the gums, to cysts in the floor of the mouth under the tongue, cysts which have a bluish colour and may grow to a large size. They are sometimes due to dilatation of the duct of the submaxillary or sublingual gland, though it is very difficult to prove this. A case dissected for us by Mr Oliver Duke, now of the Bengal army, shows very well the connection of a large ranula with Wharton's duct. At other times Wharton's duct has been found still pervious and discharging saliva, and then Rivini's duct is probably dilated. Some think mucous follicles may enlarge to produce these cysts, but Virchow throws great doubt on the existence of such follicles in this region. Phosphatic calculi are sometimes found in ranulæ, as also in similar dilatations of the pancreatic duct. Similar looking tumours of a different nature are sometimes met with here; some examples of *dermoid cysts* we have met with, one full of cholesterine and epithelium. One described by Mr Waren Tay as containing fat, like butter, was probably of the same kind. Some examples of large *lipoma* have been found here and mistaken for ranula.

The **Salivary Glands** are liable to acute inflammation in mumps. The morbid anatomy of this disease is not very pronounced; the glands soon recover their natural state. You sometimes meet with suppurative inflammation of the salivary glands, as occasionally after scarlet or continued fever. The microscope shows the suppuration to commence in the interstitial connective tissue rather than the glandular acini. Here is a specimen of calculus from the submaxillary gland, the obstruction of the duct of which, as we said, is one cause of ranula. From the same cause in Steno's duct you may have salivary fistula, all of which affections you will hear of from the surgeon. Amongst the new growths affecting these glands is the *fibro-enchondroma of the parotid.* We do not think that it is clearly made out where such disease first begins, although the glands appear more or less involved. We have already alluded to this form of tumour.

PHARYNX AND ŒSOPHAGUS

The affections of the fauces we have already spoken of, and merely place the pharynx with the œsophagus so as to include the diseases of the upper part of this tube. We must refer you back for the special diseases of the pharynx.

Malformation.—In monsters the digestive canal may be variously altered, but in otherwise normal children, deviations have been found, as the œsophagus and aorta transposed, a case of which you will find in the 'Guy's Hospital Reports.' Occasionally the œsophagus is found terminating in a *cul de sac*, or even opening into the trachea. We have met with pouches from the side of the pharynx behind the tonsil; such pouches have been described, as well as fine fistulous openings from the pharynx to the surface, representing a persistence of the fœtal branchial fissures.

We have twice seen such pouches in the œsophagus as hernial protrusion of the mucous membrane, but have never seen an accumulation of food in these pouches, though this sometimes occurs to such an extent as to compress the œsophagus and cause starvation. Such pouches are said to begin sometimes at the scar of a bronchial abscess.

Dilatation. — Cases are recorded where the œsophagus has been dilated so as to form a kind of second stomach, and with this even a process of rumination has existed. In the museum of St Thomas's there is a remarkable case of dilatation of the whole of the œsophagus. It was found accidentally after death, and unconnected with any symptoms. A similar case of yet greater dilatation of the whole œsophagus you may see here. The œsophagus widens out into a great pouch like a bird's crop. We have not seen much dilatation from stricture, for the reason, perhaps, that little or no food is taken in such cases, and the disease, which is generally cancerous, runs too rapid a course for dilatation to occur.

Contraction or Stricture is a form of disease very commonly spoken of, as if the œsophagus could be strictured in the same way as the urethra. This, however, is exceedingly rare, for in the vast majority of cases where the obstruction exists, it is due to disease within the tube, or to some tumour pressing from without; in short, generally either a cancer of the œsophagus or aneurism of the aorta. As, however, it is often certain that in the living subject such severe affections do not exist, the term stricture must still be conventionally used in those cases where there is a mere difficulty of swallowing; remembering that it is exceedingly rare to demonstrate such a condition, for during several years' observation in the post-mortem room we have never yet with met an instance of it; but we have found the muscle of the œsophagus thrice its normal thickness in heart disease, as if hypertrophied from obstruction by the pressure of the large heart. We have here, from the museum, one or two specimens which show that an ulcerative process may give rise to a cicatrisation and subsequent contraction. Here is such a constriction at the lower part of the pharynx where it joins the œsophagus; it is no doubt a contracted ulcer, and may be syphilitic. Here is one with an ulcer of a doubtful character; and this œsophagus from a boy is a very remarkable specimen, showing great thickness of the walls at its lower part; this has caused much difficulty of deglutition, and was probably due to swallowing some irritant fluid long before. Cancer we shall presently speak of.

Wounds of the pharynx and œsophagus may heal, as seen in cut throat, or as in the cases of pharyngotomy you lately saw performed by Mr Cock.

Inflammation.—*Idiopathic.*—An independent œsophagitis is probably a rare form of disease, at least it is one which is not often appreciable on the post-mortem table. We have met with small submucous abscesses apparently unimportant; but there can be no doubt that the œsophagus is sometimes affected in catarrh, as indicated by symptoms of gastric disturbance as well as difficulty of swallowing, and some cases of intense pain in swallowing have been called œsophagitis. We have already described the inflammations of the fauces; any of these, if severe, may extend down the œsophagus. Pellicular inflammation of the pharynx and œsophagus is thus sometimes met with. It may be a true *diphtheritic* inflammation proceeding from the pharynx into the œsophagus: this, however, we believe is not frequent, though we have twice observed it, but aphtha or thrush, the muguet of the French, we have several times observed to pass down the tube. A similar pellicular inflammation extending all down the œsophagus we have twice seen result from drinking boiling water; once there was pus in the mucous membrane and no pellicular layer. In conditions of extreme debility, *e.g.* advanced phthisis, prolonged low fever, &c., we have occasionally met with a circumscribed slough on the pharyngeal surface of the larynx (laryngeal part of the pharynx). But in children who have drunk boiling water, if life has been preserved for a day or two, you generally find only some lymph on the surface, and at the same time the mucous membrane abraded and shreddy in parts. If death have occurred rapidly, all you will find is serous exudation into the submucous coat, whereby the walls of the tube are thicker and softer than usual, showing the first stage of inflammation. After the swallowing of poisons the mucous membrane may become indurated or destroyed. In some cases, as by sulphuric acid, the membrane is converted into a dark brown or thick substance; if the fluid has passed down rapidly, the rugæ only are affected, and the membrane may have been destroyed and loosened in patches.

Ulcer.—We have twice met with round ulcers in the œsophagus corresponding in character with the *simple* ulcer of the stomach, and in one case associated with such a simple ulcer. We also once saw in a *syphilitic* subject two yellowish gummatous patches in the œsophagus, and once saw the tube opened by a large soft syphilitic deposit outside it.

You will see on our shelves two or three specimens showing an *opening between the œsophagus and trachea*, the remarkable feature of which is, that the aperture is circumscribed, and has no adventitious matter around it to indicate its nature; the disease not appearing cancerous, nor is there evidence of suppuration, no more being seen than a simple ulcerative process.

Perforation of the œsophagus from without is not very infrequent, you meet occasionally with the relics of bronchial glands attached to the œsophagus at the seats of old scars in its wall; thus showing bursting of old bronchial abscess into the œsophagus in childhood. Sometimes portions of necrotic vertebræ have come up from abscesses when spinal caries has opened into the œsophagus. Gangrenous cavities in the lungs or mediastinal abscesses may communicate with it, or aneurisms of the aorta burst into it.

Morbid Growths.—The most common is *cancer.* The harder *scirrhous* form of the disease is rare, but this specimen of stricture, with much thickening of the walls, appears to constitute an example of the disease. A less rare form, although not very common, is the *medullary*, as in this specimen, where you see a large encephaloid tumour growing in the œsophagus. This generally affects the lower end and may create extensive secondary cancers in the viscera. Two cases that we examined were *adenoid* in their structure, one was soft carcinoma *reticulare.* The ordinary form of cancer, however, is the *epithelial*, and this is very common. In the upper part or pharynx it may appear as a distinct circumscribed growth or tumour growing from one side, generally the anterior wall, as you see here. In most cases, however, in the upper part and always when it occurs lower down, it involves the whole circumference of the tube, and then softening down produces a large cancerous ulcer. Occasionally a great extent of the mucous surface may be affected, and the disease reaches quite into the pharynx, as you see here, where the growth has a warty appearance, and in this, where nearly the whole length of the tube is occupied by the disease in its sloughing stage. This preparation shows the pharynx destroyed by cancerous ulceration. A very frequent position for cancer is nearly opposite the bifurcation of the trachea, as you see here, where the windpipe is opened. The extension of epithelial cancer is quite local, the œsophagus and neighbouring lymphatic glands being the only parts affected, except where the disease involves parts around in contiguity with it. Thus, the cancer may reach, say, to the lung, and then, generally, breaches of its continuity occur either through lodgment of food and sloughing or through perforation by a probang, so that matters escape into the lung and form a foul sloughy pneumonic abscess. This occurs rather often and should be suspected if pneumonic symptoms arise. As a rule, we find that the destructive process advances more on the anterior than on the posterior side, for while the tube is rarely torn open on removing it from the spine, we find the front wall quite destroyed, and the disease invading fiercely the windpipe, lungs, and other parts: this position of the cancer may be accidental, owing to the less pressure in front during its growth. The surface is found sloughing, of a dark greenish colour, and very fetid; sometimes portions of dead tissue are lying loose upon it; on making a section quite through it, the adventitious tissue is seen beneath, and forming, sometimes, the sole continuation of the tube between the healthy parts. The extent of the disease is generally about three or four inches, it pervades the neighbouring glands, which are enlarged by the cancer; it often involves the pneumogastric nerves, and occasionally, *e.g.* in this specimen, the pericardium is entered; but sometimes you find only a small cancerous nodule, placed so as to obstruct the tube and cause death. Extensive cancerous ulcerations in the upper part of the canal sometimes are fatal by hæmorrhage, through perforation of the carotid; or the aorta may be opened by cancer lower down, &c.

We have twice met with general suppuration in the tissues of the neck through perforation of the pharynx or œsophagus by cancer. Although so liable to primary cancer the œsophagus may be found passing unaffected close by cancer of the lung. Tumours of the muscular tissue or *myomata* are said to have been met with in the œsophagus. Polypi, such as are found in the nose or uterus, are occasionally seen there.

Warty growths on œsophagus.—The mucous membrane of the œsophagus, as you know, is provided with papillæ, and sometimes these are increased so as to form warts, resembling warts on other parts, but more usually they consist of white flat patches, slightly raised above the surface of the mucous membrane. They consist chiefly of an increased growth of epithelium, and thus resemble the corns on the inside of the hand. They are very frequent in cases of heart disease, especially in elderly people.

Cysts in the œsophagus are met with occasionally, as in this specimen; it did not produce any symptoms. They are formed in the walls from the mucous follicles, and contain a thick mucoid fluid.

Digestive Solution.—We shall presently show you specimens of stomach dissolved by the gastric juice; and the same sometimes occurs in the œsophagus, when the contents may escape into the pleural cavity, as we have already mentioned. You will always find the contents to be highly acid under these circumstances, and the soft dissolved appearance of the part is quite characteristic; there is no sign of inflammatory reaction around, as in ulcer. You see here an œsophagus partially dissolved by the gastric juice; in a less degree you see merely the longitudinal rugæ removed or hanging in shreds. While the body is supine, the gastric contents may pass upwards into the œsophagus; and this is important to remember, for if these contents find their way into the pharynx, and then down the trachea into the bronchi, as often occurs, you might be somewhat puzzled to find such unusual matters in the lungs; the strong acidity of the contents of the trachea under these circumstances will always guide you.

Foreign Bodies.—Large masses of food may be found impacted in the pharynx or œsophagus. This great mass of meat is from the pharynx of a man who was brought in dead, suffocated by it, though it appeared within easy reach of one's fingers. Generally there is either extreme youth or softening of the pons, &c., to explain the accident, by carelessness, or paralysis of deglutition. In this œsophagus you see a large piece of pudding impacted. It killed the child, who was dead when brought to the hospital. Of late we have heard of several instances where death has been caused by the impaction of false teeth in the œsophagus. Sometimes fish bones, or pieces of bone, carelessly swallowed with meat are lodged in the œsophagus and may then ulcerate their way through, sometimes into the aorta, causing death by hæmorrhage.

PERITONEUM

Inflammation.—*Acute.*—This is characterised by the same appearances met with in other serous membranes already described, which, however, are seen in greater perfection in the peritoneum. Thus, after a few hours of inflammation, the membrane will be found reddened through its hyperæmic condition, while, at the same time, the hollow viscera are distended, their muscles having lost contractile power, so that tympanitis was produced during life. At a later period an exudation of lymph will be poured out upon it. This is often excessive, and is seen in large flakes covering all parts of the surface. In the earliest period, however, you must look carefully for the lesser signs of inflammation: thus the exudation may be so slight that it is only by scraping with a scalpel that the lymph becomes perceptible, though the dull look of the surface and its greasy feel are sufficient to detect it. The redness of the membrane is then of importance, it will be found in bands along the intestines, which bands represent the spaces where the cylindrical forms of the intestines tend to leave intervals between the coils so as to favour the lodgment of blood there by a sort of suction; on these suction bands, also, the exudation is first thrown out, while the more closely pressed surfaces have little lymph between, and very soon become coherent. As we before told you, the fluids which exude from the vessels in inflammation not only pass from the free surface, but infiltrate the membrane itself, and thus the tissue becomes softened; you will find, on examining a case of acute peritonitis, how very readily the intestines are torn; and if you take a portion, and gently incise the peritoneal coat all around, you will be able easily to separate it from the muscular layer beneath; it is important to remember this softness of an inflamed organ, it indicates the thorough change in its nutrition. The exudation may vary in kind: in acute sthenic peritonitis it is *solid lymph*, and is found covering the intestines and the abdominal parietes, and forming a layer over the liver and other organs. In other cases the peritonitis is asthenic, and the exudation is *purulent*, and then none of the signs we have mentioned are so well marked, but a quantity of milky fluid is found in the abdomen, particularly in the depending parts within the pelvis. Between these two extremes of solid and fluid there are many intermediate conditions; thus two coils of intestine may be found adherent by lymph, but when separated some fluid may be found to have collected between them. If the whole abdomen is affected, the case is generally quickly fatal; but in one remarkable case life was prolonged until the spaces between the coils of intestine were converted into abscesses, which at numerous points had burst into the canal. When the inflammation is local, it may be recovered from, or cause death more slowly.

In a case of fatal acute peritonitis you should always at once proceed to look for the cause of it; for it will be found, in most instances, to be due to a previous disease of some organ which the membrane covers. You know the peritoneum either wholly or partially envelops the various viscera in the abdomen, and therefore if active disease be progressing in these, the peritoneum is apt to become involved, so that an inflammation of it ensues. The disease of any organ may be of a slow character, so that when the peritoneum is reached it is only locally affected, and, a small amount of lymph being effused, the resulting inflammation is confined to that spot. We thus find the liver adherent to the diaphragm, while an abscess is circumscribed between them; occasionally, also, you find an abscess on the left side, which has resulted from disease of organs in that part, as, perhaps, a perforation of the stomach; also coils of intestines containing an abscess amongst them, or in the pelvis a local inflammation with similar results. If, however, an organ should suddenly rupture, and some of its natural secretions or other irritant fluid be poured out, universal and fatal peritonitis is generally set up. Thus abscess or hydatid in the liver, perforating ulcer of the stomach, perforating ulcer of the intestine (which is generally of the typhoid kind), ulcerations from old-standing organic disease of the intestine, escape of intestinal calculi from the cœcal appendix, or of any extraneous body which may have lodged there—all these are common causes. Less frequently escape of gall-stones through the ducts, rupture of ovaries, and various diseases of the genital organs; and cases are related of its occurrence from worms perforating the bowels. Owing to various local circumstances, suppuration may continue a long time behind the peritoneum without inflammation following, though a peritonitis may at last ensue. Suppuration of the kidneys very rarely penetrates to the serous membrane, and pelvic cellulitis may long continue without the peritoneum becoming involved. The extent of inflammation depends often upon the nature of the irritant which first sets it up; thus urine is one of the most irritating of fluids, and when it is extravasated into the cellular tissue, death often speedily follows: the peritoneum may then become involved without any urine having actually escaped into its cavity, as is sometimes seen after the operation of lithotomy, where an inflammation around the bladder from extravasation of urine has lit up the same disease in the peritoneum. The same results follow if an organ be ruptured by violence; and this especially when the secretion or contents of the organ are extravasated, so that bile, urine, or food, or fæcal matter escapes from its place; but it is remarkable that blood has little tendency to cause peritonitis: a large effusion of this fluid may be fatal at once; but if the organ from which it proceeds be not mortally injured, the blood does not cause severe inflammation, but undergoes changes in the peritoneum, associated with a slight effusion of plastic lymph. This comparative innocence is, no doubt, due to its having a much closer affinity to the natural secretion of the membrane than any of the other foreign products. If no local cause be found for the peritonitis, it is generally the sequel of Bright's disease.

Chronic peritonitis.—We have already said that acute inflammation of this membrane is very rarely idiopathic, but is dependent upon some anterior disease or injury of one of the organs which it covers. Chronic peritonitis, on the other hand, generally results from some morbid or altered condition of the membrane itself; although even here there is frequently some other element present to determine the inflammatory process, as tubercle or cancer. Occasionally, however, cases are met with where none of these adventitious matters can be detected, and then we are obliged to call the case one of simple chronic peritonitis. Sometimes the whole of the organs may be matted together, and thus, when the anterior abdominal walls are carefully removed, the bowels will be found united into a mass; the colon also adherent to the stomach, this to the liver, and the latter to the diaphragm; and at the same time the pelvic organs in firm union. When a slighter process of this kind has been present, there may be no exudation from the surface, but only into the substance of the membrane itself; and thus, although no adhesions may be found, the peritoneum is everywhere thickened and of an opaque white colour, the omentum also thickened, and the mesentery shortened and indurated; the surface of the liver, spleen, &c., being at the same time covered by a dense white tissue. In all such cases, however, there is probably some constitutional vice antecedent to the peritonitis.

There is a rare form of affection seen sometimes in children—an *ascites*, arising trom a chronic peritoneal affection; but we have never met with a fatal case to examine; the disease being probably strictly analogous to a chronic pleuritic effusion, and of the nature of a catarrh.

Local peritonitis.—Although the peritoneum is very ready to inflame all over when injured, yet sometimes inflammation is limited to a a greater or smaller part of the sac, either through the slightness of the irritation or through the gradual approach of a more severe irritation. A grave inflammation approaching is preceded by a wave of milder irritation at its outskirts which induces plastic products that close the peritoneum at the affected spot. Slighter irritations cause, on a surface, a plastic lymph which requires only a few days to convert it into firm and organised connective tissue. The peritoneum over irritated parts partakes of the irritation; for instance, the peritoneum over syphilitic formations in the liver, or over a liver full of miliary tubercles, or over the whole liver in persons who drink excessively, may cover itself with a plastic coat, which, if the irritation is feeble, may not adhere to parts around, as it does if more severe. It may spread down to the stomach, omentum, colon, &c. A further excitation then supervening, as through tapping, &c., may set up acute and general peritonitis.

Again, another region obnoxious to the like local inflammation is the female pelvis, where, unfortunately as it proves, the genital apparatus lies exposed in the peritoneal cavity. Through syphilitic or other irritations the internal genitalia inflame, and then you get a local peritonitis, just as about the liver, but with a greater tendency to suppurate and spread generally over the whole peritoneum. If the inflammation is slight the end is an adhesion of the parts, such as you often see in prostitutes; if greater, the inflammation may be general and fatal. Another obnoxious part is the neighbourhood of the stomach, through its liability to ulcers, which approach the surface and produce either a simple plastic peritonitis with adhesions to adjoining organs, or else, through the irritation being greater, an abscess; this bursting may produce a general peritonitis. Again, the lower end of the ileum is another of these ill-neighbourly parts; for it is here that typhoid, or, less frequently, tubercular ulceration within the bowel, brings risk of accidents just similar to those caused by ulcer of the stomach. The cœcum and its appendix, too, have a notable proneness to set up inflammation around them, and so the rectum, in a less degree. These are the chief starting-points of local peritonitis, but it may arise from inflammations of the bladder, spleen, kidney, &c., or of lymphatic glands within the abdomen, or by abscess in the abdominal wall, &c. Hernias and their treatment you will, of course, not forget. If you examine the body some time after the operation for hernia you generally find a knuckle of bowel or portion of omentum, more or less adherent, near the opening of the sac. We have twice met with a curious mass composed of the lower end of the ileum, several coils of which were adherent by ancient well-organised areolar tissue, a ball being thus formed of the size of a cocoa-nut. In one of these cases there was an old history of hernia. In the other there had been no hernia; the position suggests that the fœtal vitelline duct was concerned in the matter. In neither case had it produced any inconvenience. Over cancers you often find a soft layer of recent plastic lymph; yet it is more rare to find old adhesions. It seems as if such peritonitis is often a part of the final break up. Indeed, you should notice that often a degree of peritonitis seems to start up in the dependent part of the peritoneum, in cases of extreme exhaustion from very chronic disease. Thus, we have found several times, under these circumstances, a little pus in the lowest part of the pelvic peritoneum, or in its subserous tissue, or in the bottom of a harmless hernial sac. It appears as though the same thing occurs in the peritoneum, as in the bronchial-tubes, &c. in long-lasting moribund states, that is, the free secretion corrupts and becomes an irritant to the surface in contact. Such a cadaveric peritonitis of dependent parts we have seen not unfrequently.

You must notice how the peritoneum is made to share other troubles of organs it covers; we shall see that in cancer of the stomach germs escape into the peritoneum and fall down to establish themselves in the pelvis. No doubt, generally, when the local drainage is overwhelmed by excess of diseased products, the peritoneum suffers from the soakage of these products for which it offers a free course.

We have seen inflammatory effusion limited to the lesser bag of the peritoneum so as to form a large cyst in a case of injury with fracture of the pancreas, and in a case of old hydatid abscess.

The old adhesions that result from local peritonitis are as common as pleuritic adhesion, but they are apt to be attended with more serious consequences. In many parts they are of no importance, as, for instance, when they connect the liver with the diaphragm; but by constricting the intestines such bands may give rise to fatal results, as you see in some of these specimens; a band of adhesion passes down over a portion of bowel and directly constricts it; or joined by its end to some other part, it may produce a loop through which a coil of bowel may slip. Again, it not uncommonly happens that the cœcal appendix or a cœcal diverticulum of the ileum becomes adherent at its end and forms a loop through which intestine may pass. Again, other organs, besides the intestine, may suffer, as when the chronic local inflammation causes adhesions of Fallopian tubes, ovaries, &c. In several cases of extra-uterine fœtation which we have seen the omentum has been adherent to the broad ligament, as though its union had disturbed the action of the Fallopian tube. Still it is especially as to the intestine that these adhesions are important.

You may often find in fatal jaundice a union of the several organs about the region of the gall-bladder, as the colon, duodenum, and gall-bladder with its ducts, all firmly united by dense fibrous tissue; on cutting them apart, a chronic local inflammation is seen to have occurred, and the cause of all is probably a gall-stone which may be found in the bladder or ducts.

Hæmorrhagic peritonitis.—We have already spoken of blood being found mixed with the inflammatory exudations in pericarditis and pleuritis, and now you must notice that the same sometimes occurs in the peritoneum. On first opening the abdomen you are surprised to see effused blood covering the intestines and other parts; but on closer examination you discover that it is merely lymph coloured by blood, having the same origin as the hæmorrhagic pleurisy before mentioned.

Tubercular peritonitis.—This is not so frequent as appears to be supposed. It would excite your surprise if you examined many cases of pulmonary phthisis to see tubercular ulceration in the bowels, so extensive as it often is, and yet not once in a hundred times does it light up tubercular peritonitis, though, as we shall see, it nearly always affects the sub-peritoneal tissue. Tubercular peritonitis occurs in a rather different class of cases, and it is generally a primary affection. It is apt to ally itself with tubercular pleurisy and pericarditis, and you may find all the serous membranes thus affected when the parenchymatous organs escape. Generally, however, there is tubercle elsewhere, although the tubercle of the peritoneum is the main disease. It appears under at least three different forms. The first may be called tubercular ascites; the surface is free from adhesions in the whole or nearly the whole of its extent. The tubercles form small knots of sizes from that of dust to swan-shot; they are thickest on the under surface of the diaphragm, the intestine suffering less. In these cases there is generally a great quantity of more or less turbid liquid effusion in the cavity, and clinically they are of the nature of inflammatory peritoneal dropsy. This form of disease is very like cancerous peritonitis, which we shall presently describe to you. Sometimes you can scarcely tell whether the little knots are tubercular or cancerous in their nature. At other times they are quite certainly tuberculous; in no other place do you see tubercle and cancer so closely approach each other in likeness, except, perhaps, in the lymphatic glands.

The second form, also rather rare, is latent peritoneal tubercle; such cases are clinically very obscure. The patient wastes away, and has some indefinite local abdominal symptoms, say vesical irritation or irregular annoyance of the alimentary canal; treatment may be directed to the bladder or to symptoms resembling ileus, but after death one finds the peritoneum studded with tubercles in the midst of a soft semi-organized pellucid matter by which its surfaces adhere. The local irritation is then found to be due to the casual collection of a mass of lymph about the bladder or rectum or other suffering part.

But the third form is the most frequent and that which is best understood as tubercular peritonitis. In fatal cases of this kind you will find the intestines inextricably united together, so that any attempt to separate them will only end in their laceration and rupture; the abdominal walls are adherent both to the intestine and to the liver, from which often they have to be cut, as well as from all the remaining viscera. The intestines become united, and the disease still continuing, ulcers form where the tubercular matter accumulates between the coils and the intestines are perforated, so that in the firmly united mass of intestine, many coils are found directly communicating with one another by perforations. Occasionally the coils are adherent around considerable spaces full of tuberculous pus which forms between them; into these spaces the contents of the bowels pass, and a local fæcal abscess is produced. When in this manner such perforations open into a free space, they present a peculiar appearance, owing to the contraction of the muscular coat, and the mucous membrane becoming everted, so that instead of exhibiting a small round opening, you perceive a raised swollen nodule like a rosebud, as in this preparation. More rarely such fæcal abscesses attempt to make their way through the abdominal parietes, and perhaps burst or are opened, producing a fæcal fistula.

Cancerous peritonitis.—There is another form of peritonitis, where the morbific cause is cancer. The characters of the disease closely resemble the first form of tubercular peritonitis, the liquid products being more profuse, while the lymph on the surface is less in amount, the intestines being free, so that the case is often clinically a simple ascites; many of them are mistaken for hepatic dropsy. The disease is remarkable, also, in its being generally local, other parts of the body may not be affected with cancer, nor even, often, the abdominal organs themselves. It is this which renders the disease so obscure during life. The sole symptom generally being ascites, it is only in the absence of other causes for the dropsy that a suspicion is created as to its nature; but if the fluid be drawn off by tapping it is generally found tinged with altered blood, so as to be brownish or purplish in colour. This, as in the liquid of a hydrothorax, is characteristic of cancer. It is a form of disease frequent enough, though not much described, but it was always pointed out by Dr Addison in his lectures. On opening the abdomen, a quantity of slightly bloody serum is found, and the peritoneum is seen covered with a number of hard tubercles, the omentum infiltrated with the same products, and the mesentery hardened and contracted. The thickening of the latter parts is often very remarkable, so that they form solid masses an inch or two thick, the omentum is drawn up to the colon, forming a hard ridge beneath it, which should be felt for during life in doubtful cases. Hard nodules are sprinkled over the intestines and abdominal walls, chiefly the under surface of the diaphragm and the flanks; these nodules on microscopic examination, are often found to consist of fibres with a few spindle cells, so that the disease might be called fibroma, and might be thought of in connection with keloid rather than cancer; the disease is called peritonitis because of the thickening and contraction of the serous membrane showing that a chronic inflammation has been going on; the same association of new growth with inflammation which we see in tubercular peritonitis, phthisis, &c.; likewise, as in such cases, generally, it is still a question how far the new formation is necessarily antecedent to the inflammatory changes, so in the present instance the same difficulty occurs. There can be no doubt, however, that in all such instances the process is one, in great measure, of chronic inflammation, but peculiar, owing to unknown circumstances; and, in the present case, putting on a cancerous form, as it does tuberculous in others. A quasi-inflammatory origin of this kind of cancer is often strongly suggested through its arising secondarily from cancers of the sigmoid colon, or ovary, stomach, &c., as though the cancerous action had spread from these parts swiftly over the whole peritoneum in a way that can only be compared to inflammatory activity; we have sometimes found cancerous knots in bands of old false membrane in the peritoneum. On the other hand, cancerous peritonitis may dip into and invade the intestine.

Morbid Growths.—*Cancer* frequently involves the peritoneum when it affects any viscus beneath and reaches the peritoneal surface. The cancer germs may then be cast into the peritoneal cavity, and without exciting a general cancerous peritonitis may settle down to the bottom of the cavity about the pelvis, there setting up cancer in the form of little knots, which are called "daughter tubercles" by Virchow in relation to the mother tubercles they came from.

Tubercle may be often found in the peritoneum in cases of general tuberculosis, especially in the omentum, when the miliary tubercles often accompany the vessels, but its especial importance arises when it is primary and accompanied by an inflammatory process, as already described.

Colloid.—This kind of cancer is most frequently met with in the abdomen. Its general seat is in the neighbourhood of the stomach, colon, and omentum; or it is connected with a similar disease of the ovary. In the slighter cases it occurs merely within the walls of the stomach, &c., as will be mentioned under the several organs, but it shows itself apt to spread in the serous membrane. Thus the peritoneum may sometimes be found covered with these gelatinous masses growing from its surface, and the omentum immensely thickened, and converted into a long, flattened, hard tumour. A section of this would show the interior to be white, and having somewhat of a honeycomb appearance, consisting of a fibrous network containing the colloid matter within it. Sometimes this has formed in such large quantities, that when the abdomen is opened several pints of it have poured out. In these cases its seat appears to be the subserous coat, involving also the peritoneum, and the walls of the hollow organs, as the stomach. In this beautiful and rare specimen, the liver is implicated in the disease. When examined, you find it composed of a network of delicate fibres, somewhat like the tissue of the lung, only finer and softer, and the spaces larger. In these spaces or loculi, the white transparent gum-like colloid matter is contained. The microscope shows cells or nuclei in its midst which much resemble mucous corpuscles, but where the structure is firmer the spaces may be found filled with clusters of cells having all the ordinary epitheloid characters of cancer cells; all gradations from these cells into the mucous matter by "mucoid degeneration" may be met with.

Morbid Contents.—*Blood.*—This is constantly found in the abdomen as a result of rupture of an aneurism, or of injury to the liver, spleen, &c.; we have seen seven ounces result from bleeding of an hepatic cancer. The blood may be mixed, as we have just said, with inflammatory lymph. It is often a question of great importance to ascertain the extent of hæmorrhage which will prove fatal. The bleeding into the abdomen in cases of aneurism affords an opportunity of measuring the fatal loss. We have found generally four to five pints, once only three. But in a case of aneurism bursting into the thorax we once found seven pints of blood; there was, however, here reason to think that the hæmorrhage had occurred on two or three occasions, when a much larger amount of bleeding can be borne than at once.

Where the amount of blood effused has not been sufficient to cause death, little or no peritonitis follows, but various changes take place in the blood; the fluid matter is absorbed, the fibrinous part adheres to the serous membrane, and the colouring matter undergoes changes to a darker colour. In the brain, you remember, it becomes lighter. On the intestine, however, it becomes very dark, and in time almost black. In rupture of the liver or spleen, if veins be opened, the blood is portal and of a pitchy character.

A *black pigment* is often met with in the peritoneum, especially in that lining the pelvis; there is then generally some evidence of a former peritonitis; and as in inflammation some blood is often found effused with the lymph, it is probable that in these cases the source of the colouring matter is hæmatine. Occasionally the intestines are found uniformly black, at other times, spotted. Such a condition sometimes occurs in connection with the deposition of pigment in other parts.

Air.—We have already alluded to the subject of air in the serous cavities as the result of secretion or decomposition. We have never seen it in the pleura, unless with clear evidence of its entry from without, and never an undoubted instance of it in the pericardium. In the peritoneum it is frequently found after perforation of the intestine, when the gas has clearly escaped into the cavity. In one case, which occurred here about a year ago, it was evident that gas had existed in the abdomen for two or three weeks before death. The abdomen fluctuated, showing fluid, but at the same time was boggy and tympanitic, and on shaking the patient a splash was heard. On post-mortem examination, a similar sound was heard on succussion; and on cutting through the walls a gush of air came out, the intestines were found contracted at the posterior part, and covered with lymph in which blood was mingled, as just now described. The source of the air was not satisfactorily made out, whether a decomposition of the blood and other products of effusion, or whether there had been an escape from the intestine; it is very likely that the latter was the cause, though, from the subsequent matting together of the intestine, it was impossible to make it out.

We have seen cases where *chyle* was found in the abdominal cavity from obstruction of thoracic or lacteal ducts. One of these cases you will find recorded in our *Reports*.

Loose bodies.—These may be of various kinds, as *biliary* or *intestinal calculi*, which may have escaped from their respective canals; it is said also that a loose *uterine tumour* has been found, and this we can well believe, from a case we once saw where a large tumour hung by the thinnest possible pedicle. In most instances, however, the loose bodies are small and of a definite character, and have, no doubt, one source. They are oval or round and flattened, resembling a bean, enclosed in a capsule, and within composed of fibrous tissue and fat. You will see several specimens in these bottles, some loose and others attached. Some of these were taken by Dr Burton Brown from bodies in the dissecting-room, and some you see in course of development in the appendices epiploicæ; in fact, these latter processes become altered in structure and then drop off, constituting the loose tumours. One such body is described by a surgeon as being found in a hernial sac when he proceeded to operate.

Hydatids are sometimes found loose in the abdomen, or are developed between adherent organs; one, between stomach, colon, and liver, was remarkable, as the man fell twenty-five feet and broke his pelvis, yet did not burst the large hydatid.

STOMACH

Malformation.—The stomach may be absent in unviable abortions, or it may be congenitally small. Some cases of hour-glass contraction are congenital. The stomach may be congenitally misplaced in the thorax through an opening in the diaphragm.

Dilatation.—This is generally a secondary affection, and is seen especially in cases of obstruction of the pyloric end. Just as the stomach contracts when little food enters from disease of the cardiac end, so it dilates in cases of scirrhous pylorus. In some cases of this affection which we have examined, on opening the abdomen the stomach was the only organ seen, being immensely distended and reaching to the pelvis; and you may sometimes observe in the living subject the outline of the stomach thus distended from the great accumulation of fluid within it. Sometimes the distension is of a paralytic nature; and such paralysis we have twice known prove fatal after surgical operations. Sometimes the cause of the paralysis could not be found, but in one case where the dorsal vertebræ from the fourth to the tenth were involved in a carious abscess which implicated the splanchnic nerves, the distension was immense; we shall presently speak of general distension of the intestines from diseases implicating the solar plexus.

Contraction.—When little food enters the stomach it shrinks of course towards its œsophageal attachment, hence when gastrotomy is performed for stricture of the œsophagus the stomach is generally opened much nearer the pylorus than you would expect. A similar contraction too may sometimes be seen where cancer is diffused through the coats, or when there is extensive ulceration and thickening of the walls. In these two preparation-bottles you will see stomachs in the state called hourglass contraction. You see both organs appear as if a ligature had been passed around their middle, dividing them into two portions;

in one the peritoneal coat is thickened, which, no doubt, denotes the site of a healed chronic ulcer; at least this was the case in the two or three examples we have met with on the dead body.

Injury.—Wounds of the stomach leading to evacuation of its contents are generally fatal. Remarkable exceptional cases, however, occur, where the wound becomes adherent to the abdominal walls, and closes; and also where the edges unite with an external wound, and a permanent fistula remains, as in the well-known case of Alexis St Martin and a few other recorded examples. Most of you saw a man lately in the hospital, in whom there was little doubt that the stomach had been wounded by a stab with a knife, and yet he recovered; also a similar one which occurred at the same time elsewhere, in which it was almost certain that the stomach had been opened. We met with another where the stomach was torn through for an inch length over the spinal column in a man who was run over. He lived two weeks and then died of double pleurisy, but the rent in the stomach was adherent to the liver and undergoing cure. In all the cases where the organ has been opened by the surgeon to introduce nourishment, death has soon taken place. Sometimes you see a partial laceration of the coats of the stomach, as in this example, where the mucous membrane is detached and lacerated, a long rent being seen on its inner surface. It came from a boy who was run over. We have also on our shelves a stomach with a laceration, which is supposed to have occured during the act of vomiting; but this, we think, is very doubtful. The stomach may be displaced into the thorax through a ruptured diaphragm, producing *diaphragmatic hernia.* This can only occur on the left side, when the organ with some of the intestines may escape into the chest. In some instances, where there has been no history of accident, it has been thought that it might have arisen from disease; in this specimen the misplacement was congenital.

Congestion.—We do not know any more important morbid condition of the stomach than congestion, for it is only by a proper knowledge of this and similar states that you will be able to recognise the results of poisoning by irritant substances; and even after long observation you will often find it very difficult to pronounce in any case whether the organ be inflamed or not, seeing that the ordinary post-mortem appearances are so varied, according to the modes of death and conditions of the organs, especially the presence or absence of food. What makes the difficulty greater is, that congestion and inflammation pass into one another, and that the latter is probably more often present than is usually believed The example we particularly wish to bring before you is the stomach in heart disease, which will be frequently shown you in the post-mortem room. If the cardiac affection be of an obstructive kind, so that there is great congestion in all the organs of the body, the mucous membrane of the stomach is affected in common with other structures, and consequently presents a very remarkable appearance, and one which would be more likely to be regarded as acute inflammation than many cases of the action of irritant poisons. The interior is of a dark red or purple colour throughout, and on close examination this is found to be due to a hyperæmic state of the vessels, or true capillary injection; and, besides this, there is a large mucous secretion from the interior of the stomach, the whole being sometimes found covered with mucus of so tenacious a character that a strong stream of water is barely sufficient to wash it off; there is also sometimes an effusion in the mucous membrane itself, and in the submucous tissue, and then the whole coats appear thickened, and the organ feels fleshy and heavy instead of membranous: when you remember the epigastric pain, sickness, &c., sometimes attending heart disease, and then look upon such a condition as this which is found after death, you can hardly doubt that this extreme congestion has actually passed into an inflammation; the ulceration which is sometimes found in conjunction with it we will presently mention. Thus the stomach in heart disease approaches much more nearly to an inflammation than many cases where an irritant poison has been swallowed; indeed we have never seen any case of the latter which at all equalled it, and, therefore, we should warn you not to pronounce too hastily that an irritant poison has been taken through any amount of redness or of mucous secretion that is found; for we have never yet met with a case where any such condition could have warranted the suggestion of such a poison unless the fact had been previously known; indeed we believe it is only when corrosion has been produced that you can speak with certainty of the action of poison from the anatomical state of the stomach; a chemical study of the contents is always essential.

Gastritis.—*Acute catarrhal.*—It is remarkable how opinions have varied, and do still, with reference to the existence or frequency of gastritis. Formerly, when morbid anatomy was less cultivated, the various symptoms denoting severe stomach derangements were attributed to inflammation, and gastritis was one of the most frequently recognised disorders. Subsequently, however, when the viscera were more examined, and some tangible or very visible alterations were required to prove the existence of a morbid process, gastritis came into disrepute, and some eminent pathologists have denied altogether the existence of such a disease; failing, more especially, to find alterations in the stomach of drunkards, and others, where inflammations had been thought so often to occur. It is probable, however, that this opinion was due to the absence of suppuration, and such well-marked results of inflammation, and the non-recognition of minute changes in the mucous membrane. Thus the membrane in such cases is found to show a milky opacity, while it is soft and thick, so that it is more lacerable than natural. Its state microscopically shows very little that is characteristic. The epithelium of the follicles is swollen and cloudy, and may fall out coherent like renal tube casts. If the inflammation be severe, lymphoid cells of an inflammatory nature may be found between and under or even inside the tubes. Although the structural evidence is weak there can be little doubt that acute gastritis is a frequent affection. Thus the stomach in cardiac disease already mentioned, shows a *sub-acute inflammation*, as evidenced by the hyperæmic condition of the blood-vessels and the mucous secretion; and such a state, though less marked, may be often met with in other forms of disease, as in morbus Brightii, &c.; and in cases of Addison's disease, where great irritability of stomach has existed, some evidence of a gastritis has also been present. If we notice the symptoms attending such cases as these, we cannot do otherwise than suppose an inflammation is present. The frequency of stomach derangement in common catarrh, where the bronchi are thought to be the parts only involved, would suggest it as a not uncommon complaint; we have often found in cases of acute pneumonia and other febrile diseases, the stomach in a state of minute reddening, its surface coated with tenacious and sometimes semi-opaque mucus, different in character from the natural clear mucus of the stomach walls, and in great excess of the normal quantity; this corresponds with the loss of appetite which is experienced in such febrile states. When the microscope cannot be used, it is rather by these products of the inflammation than by any redness that we should be guided in judging of a catarrhal inflammation, for inflammatory redness of the stomach or of the skin may quite disappear after death; we would advise you always to examine the stomach, and become well acquainted with its morbid appearances.

Phlegmonous gastritis, or suppurative inflammation of the stomach wall is rare; we have met with two instances in the form of sub-mucous abscesses, some of them opening into the cavity and associated with suppuration in the track of the portal vein. In one of these the rectum showed similar abscesses. Although this is a rare disease, yet thirty cases were collected by Ackermann, who found that puerperal pyæmia appeared to be its more frequent cause; the state in one case was compared by Virchow to carbuncle. It is apt to set up pyæmic abscess of the liver, which is a rare result of chronic gastric ulcer.

Croupous gastritis.—This is, perhaps, still more rare; it has been seen by Sir W. Jenner in diphtheria. Dr Delafield says, "it is almost always secondary to typhus, pyæmia, puerperal fever, cholera, dysentery, the exanthemata and irritating poisons. The false membrane is in small patches, and may line a large part of the stomach." He gives a case of idiopathic croupous gastritis, in a man of forty-six, fatal in eight days. Dr Wilson Fox has seen it in phthisis. We have seen it in membrane into this form. This is probably the cause, for we think it is not yet satisfactorily proved that it is due to adventitious structure, or is really morbid—at least not more so than the analogous case of *cutis anserina*. If you examine sections of the inflamed stomach hardened in chromic acid, you will have no difficulty in seeing that important changes have taken place, although these do not usually amount to a disintegration of the structure. Thus you find the interstitial tissue between the tubes thickened in places; if you examine patches of such thickening you will find that the apparently new matter is made up largely of shrivelled tubes. At the same time the tubes become much less straight and parallel than they properly are, so that they look confused, and it is difficult to get sections of their whole length; their walls are thick, their contents are fatty and granular in many parts, and in some places quite disintegrated. Sometimes the mouths of the tubes are blocked up, and the deeper parts dilated into cysts. The condition of the solitary glands is not constant, sometimes they are large and ulcerated, sometimes they are wasted so as to be scarcely discoverable. We have not seen lymphoid tissue between the glands such as some describe in chronic gastritis. The whole of the changes correspond with the conditions observed in glandular organs, especially the kidney, in chronic inflammation, indeed chronic gastritis is often associated with granular kidney and similar chronic changes, just as acute gastritis is associated with acute pneumonia and other febrile disorders. Since Drs Habershon and Handfield Jones first described this condition, much attention has been given to it, especially by Dr Wilson Fox, in very able researches; our own results correspond very nearly with those of these authors, but we have been much impeded by the unfortunate difficulty in obtaining good clinical evidence of the gastritis. Often you will find much change where there were few symptoms, and little change where the symptoms led you to expect much.

The mucous membrane of the stomach may be found affected by lardaceous disease, it then looks sodden and dull, and its surface is stained of a deep walnut colour by iodine. This condition is only found as a part of a very general lardaceous disease of the viscera, and as in other parts, the disease begins in the small arteries. This state produces a liability to catarrh, as in the kidney and elsewhere.

Ulceration.—*Catarrhal.*—Ulceration, as an effect of inflammation, is not so common as might be thought; ulceration being generally cancerous, or else of that equally characteristic kind known as "chronic ulcer." It may occur, however, from the effects of poison, as we have seen, and sometimes may arise as scattered minute ulcerations in connection with acute catarrhal gastritis. Such a specimen is here, where minute ulcerations are seen on the stomach.

may not be affected with cancer, nor even, often, the abdominal organs themselves. It is this which renders the disease so obscure during life. The sole symptom generally being ascites, it is only in the absence of other causes for the dropsy that a suspicion is created as to its nature; but if the fluid be drawn off by tapping it is generally found tinged with altered blood, so as to be brownish or purplish in colour. This, as in the liquid of a hydrothorax, is characteristic of cancer. It is a form of disease frequent enough, though not much described, but it was always pointed out by Dr Addison in his lectures. On opening the abdomen, a quantity of slightly bloody serum is found, and the peritoneum is seen covered with a number of hard tubercles, the omentum infiltrated with the same products, and the mesentery hardened and contracted. The thickening of the latter parts is often very remarkable, so that they form solid masses an inch or two thick, the omentum is drawn up to the colon, forming a hard ridge beneath it, which should be felt for during life in doubtful cases. Hard nodules are sprinkled over the intestines and abdominal walls, chiefly the under surface of the diaphragm and the flanks; these nodules on microscopic examination, are often found to consist of fibres with a few spindle cells, so that the disease might be called fibroma, and might be thought of in connection with keloid rather than cancer; the disease is called peritonitis because of the thickening and contraction of the serous membrane showing that a chronic inflammation has been going on; the same association of new growth with inflammation which we see in tubercular peritonitis, phthisis, &c.; likewise, as in such cases, generally, it is still a question how far the new formation is necessarily antecedent to the inflammatory changes, so in the present instance the same difficulty occurs. There can be no doubt, however, that in all such instances the process is one, in great measure, of chronic inflammation, but peculiar, owing to unknown circumstances; and, in the present case, putting on a cancerous form, as it does tuberculous in others. A quasi-inflammatory origin of this kind of cancer is often strongly suggested through its arising secondarily from cancers of the sigmoid colon, or ovary, stomach, &c., as though the cancerous action had spread from these parts swiftly over the whole peritoneum in a way that can only be compared to inflammatory activity; we have sometimes found cancerous knots in bands of old false membrane in the peritoneum. On the other hand, cancerous peritonitis may dip into and invade the intestine.

Morbid Growths.—*Cancer* frequently involves the peritoneum when it affects any viscus beneath and reaches the peritoneal surface. The cancer germs may then be cast into the peritoneal cavity, and without exciting a general cancerous peritonitis may settle down to the bottom of the cavity about the pelvis, there setting up cancer in the form of little knots, which are called "daughter tubercles" by Virchow in relation to the mother tubercles they came from.

Tubercle may be often found in the peritoneum in cases of general tuberculosis, especially in the omentum, when the miliary tubercles often accompany the vessels, but its especial importance arises when it is primary and accompanied by an inflammatory process, as already described.

Colloid.—This kind of cancer is most frequently met with in the abdomen. Its general seat is in the neighbourhood of the stomach, colon, and omentum; or it is connected with a similar disease of the ovary. In the slighter cases it occurs merely within the walls of the stomach, &c., as will be mentioned under the several organs, but it shows itself apt to spread in the serous membrane. Thus the peritoneum may sometimes be found covered with these gelatinous masses growing from its surface, and the omentum immensely thickened, and converted into a long, flattened, hard tumour. A section of this would show the interior to be white, and having somewhat of a honeycomb appearance, consisting of a fibrous network containing the colloid matter within it. Sometimes this has formed in such large quantities, that when the abdomen is opened several pints of it have poured out. In these cases its seat appears to be the subserous coat, involving also the peritoneum, and the walls of the hollow organs, as the stomach. In this beautiful and rare specimen, the liver is implicated in the disease. When examined, you find it composed of a network of delicate fibres, somewhat like the tissue of the lung, only finer and softer, and the spaces larger. In these spaces or loculi, the white transparent gum-like colloid matter is contained. The microscope shows cells or nuclei in its midst which much resemble mucous corpuscles, but where the structure is firmer the spaces may be found filled with clusters of cells having all the ordinary epitheloid characters of cancer cells; all gradations from these cells into the mucous matter by "mucoid degeneration" may be met with.

Morbid Contents.—*Blood.*—This is constantly found in the abdomen as a result of rupture of an aneurism, or of injury to the liver, spleen, &c.; we have seen seven ounces result from bleeding of an hepatic cancer. The blood may be mixed, as we have just said, with inflammatory lymph. It is often a question of great importance to ascertain the extent of hæmorrhage which will prove fatal. The bleeding into the abdomen in cases of aneurism affords an opportunity of measuring the fatal loss. We have found generally four to five pints, once only three. But in a case of aneurism bursting into the thorax we once found seven pints of blood; there was, however, here reason to think that the hæmorrhage had occurred on two or three occasions, when a much larger amount of bleeding can be borne than at once.

Where the amount of blood effused has not been sufficient to cause death, little or no peritonitis follows, but various changes take place in the blood; the fluid matter is absorbed, the fibrinous part adheres to the serous membrane, and the colouring matter undergoes changes to a darker colour. In the brain, you remember, it becomes lighter. On the intestine, however, it becomes very dark, and in time almost black. In rupture of the liver or spleen, if veins be opened, the blood is portal and of a pitchy character.

A *black pigment* is often met with in the peritoneum, especially in that lining the pelvis; there is then generally some evidence of a former peritonitis; and as in inflammation some blood is often found effused with the lymph, it is probable that in these cases the source of the colouring matter is hæmatine. Occasionally the intestines are found uniformly black, at other times, spotted. Such a condition sometimes occurs in connection with the deposition of pigment in other parts.

Air.—We have already alluded to the subject of air in the serous cavities as the result of secretion or decomposition. We have never seen it in the pleura, unless with clear evidence of its entry from without, and never an undoubted instance of it in the pericardium. In the peritoneum it is frequently found after perforation of the intestine, when the gas has clearly escaped into the cavity. In one case, which occurred here about a year ago, it was evident that gas had existed in the abdomen for two or three weeks before death. The abdomen fluctuated, showing fluid, but at the same time was boggy and tympanitic, and on shaking the patient a splash was heard. On post-mortem examination, a similar sound was heard on succussion; and on cutting through the walls a gush of air came out, the intestines were found contracted at the posterior part, and covered with lymph in which blood was mingled, as just now described. The source of the air was not satisfactorily made out, whether a decomposition of the blood and other products of effusion, or whether there had been an escape from the intestine; it is very likely that the latter was the cause, though, from the subsequent matting together of the intestine, it was impossible to make it out.

We have seen cases where *chyle* was found in the abdominal cavity from obstruction of thoracic or lacteal ducts. One of these cases you will find recorded in our *Reports.*

Loose bodies.—These may be of various kinds, as *biliary* or *intestinal calculi*, which may have escaped from their respective canals; it is said also that a loose *uterine tumour* has been found, and this we can well believe, from a case we once saw where a large tumour hung by the thinnest possible pedicle. In most instances, however, the loose bodies are small and of a definite character, and have, no doubt, one source. They are oval or round and flattened, resembling a bean, enclosed in a capsule, and within composed of fibrous tissue and fat. You will see several specimens in these bottles, some loose and others attached. Some of these were taken by Dr Burton Brown from bodies in the dissecting-room, and some you see in course of development in the appendices epiploicæ; in fact, these latter processes become altered in structure and then drop off, constituting the loose tumours. One such body is described by a surgeon as being found in a hernial sac when he proceeded to operate.

Hydatids are sometimes found loose in the abdomen, or are developed between adherent organs; one, between stomach, colon, and liver, was remarkable, as the man fell twenty-five feet and broke his pelvis, yet did not burst the large hydatid.

STOMACH

Malformation.—The stomach may be absent in unviable abortions, or it may be congenitally small. Some cases of hour-glass contraction are congenital. The stomach may be congenitally misplaced in the thorax through an opening in the diaphragm.

Dilatation.—This is generally a secondary affection, and is seen especially in cases of obstruction of the pyloric end. Just as the stomach contracts when little food enters from disease of the cardiac end, so it dilates in cases of scirrhous pylorus. In some cases of this affection which we have examined, on opening the abdomen the stomach was the only organ seen, being immensely distended and reaching to the pelvis; and you may sometimes observe in the living subject the outline of the stomach thus distended from the great accumulation of fluid within it. Sometimes the distension is of a paralytic nature; and such paralysis we have twice known prove fatal after surgical operations. Sometimes the cause of the paralysis could not be found, but in one case where the dorsal vertebræ from the fourth to the tenth were involved in a carious abscess which implicated the splanchnic nerves, the distension was immense; we shall presently speak of general distension of the intestines from diseases implicating the solar plexus.

Contraction.—When little food enters the stomach it shrinks of course towards its œsophageal attachment, hence when gastrotomy is performed for stricture of the œsophagus the stomach is generally opened much nearer the pylorus than you would expect. A similar contraction too may sometimes be seen where cancer is diffused through the coats, or when there is extensive ulceration and thickening of the walls. In these two preparation-bottles you will see stomachs in the state called hourglass contraction. You see both organs appear as if a ligature had been passed around their middle, dividing them into two portions;

in one the peritoneal coat is thickened, which, no doubt, denotes the site of a healed chronic ulcer; at least this was the case in the two or three examples we have met with on the dead body.

Injury.—Wounds of the stomach leading to evacuation of its contents are generally fatal. Remarkable exceptional cases, however, occur, where the wound becomes adherent to the abdominal walls, and closes; and also where the edges unite with an external wound, and a permanent fistula remains, as in the well-known case of Alexis St Martin and a few other recorded examples. Most of you saw a man lately in the hospital, in whom there was little doubt that the stomach had been wounded by a stab with a knife, and yet he recovered; also a similar one which occurred at the same time elsewhere, in which it was almost certain that the stomach had been opened. We met with another where the stomach was torn through for an inch length over the spinal column in a man who was run over. He lived two weeks and then died of double pleurisy, but the rent in the stomach was adherent to the liver and undergoing cure. In all the cases where the organ has been opened by the surgeon to introduce nourishment, death has soon taken place. Sometimes you see a partial laceration of the coats of the stomach, as in this example, where the mucous membrane is detached and lacerated, a long rent being seen on its inner surface. It came from a boy who was run over. We have also on our shelves a stomach with a laceration, which is supposed to have occured during the act of vomiting; but this, we think, is very doubtful. The stomach may be displaced into the thorax through a ruptured diaphragm, producing *diaphragmatic hernia*. This can only occur on the left side, when the organ with some of the intestines may escape into the chest. In some instances, where there has been no history of accident, it has been thought that it might have arisen from disease; in this specimen the misplacement was congenital.

Congestion.—We do not know any more important morbid condition of the stomach than congestion, for it is only by a proper knowledge of this and similar states that you will be able to recognise the results of poisoning by irritant substances; and even after long observation you will often find it very difficult to pronounce in any case whether the organ be inflamed or not, seeing that the ordinary post-mortem appearances are so varied, according to the modes of death and conditions of the organs, especially the presence or absence of food. What makes the difficulty greater is, that congestion and inflammation pass into one another, and that the latter is probably more often present than is usually believed The example we particularly wish to bring before you is the stomach in heart disease, which will be frequently shown you in the post-mortem room. If the cardiac affection be of an obstructive kind, so that there is great congestion in all the organs of the body, the mucous membrane of the stomach is affected in common with other structures, and consequently presents a very remarkable appearance, and one which would be more likely to be regarded as acute inflammation than many cases of the action of irritant poisons. The interior is of a dark red or purple colour throughout, and on close examination this is found to be due to a hyperæmic state of the vessels, or true capillary injection; and, besides this, there is a large mucous secretion from the interior of the stomach, the whole being sometimes found covered with mucus of so tenacious a character that a strong stream of water is barely sufficient to wash it off; there is also sometimes an effusion in the mucous membrane itself, and in the submucous tissue, and then the whole coats appear thickened, and the organ feels fleshy and heavy instead of membranous: when you remember the epigastric pain, sickness, &c., sometimes attending heart disease, and then look upon such a condition as this which is found after death, you can hardly doubt that this extreme congestion has actually passed into an inflammation; the ulceration which is sometimes found in conjunction with it we will presently mention. Thus the stomach in heart disease approaches much more nearly to an inflammation than many cases where an irritant poison has been swallowed; indeed we have never seen any case of the latter which at all equalled it, and, therefore, we should warn you not to pronounce too hastily that an irritant poison has been taken through any amount of redness or of mucous secretion that is found; for we have never yet met with a case where any such condition could have warranted the suggestion of such a poison unless the fact had been previously known; indeed we believe it is only when corrosion has been produced that you can speak with certainty of the action of poison from the anatomical state of the stomach; a chemical study of the contents is always essential.

Gastritis.—*Acute catarrhal.*—It is remarkable how opinions have varied, and do still, with reference to the existence or frequency of gastritis. Formerly, when morbid anatomy was less cultivated, the various symptoms denoting severe stomach derangements were attributed to inflammation, and gastritis was one of the most frequently recognised disorders. Subsequently, however, when the viscera were more examined, and some tangible or very visible alterations were required to prove the existence of a morbid process, gastritis came into disrepute, and some eminent pathologists have denied altogether the existence of such a disease; failing, more especially, to find alterations in the stomach of drunkards, and others, where inflammations had been thought so often to occur. It is probable, however, that this opinion was due to the absence of suppuration, and such well-marked results of inflammation, and the non-recognition of minute changes in the mucous membrane. Thus the membrane in such cases is found to show a milky opacity, while it is soft and thick, so that it is more lacerable than natural. Its state microscopically shows very little that is characteristic. The epithelium of the follicles is swollen and cloudy, and may fall out coherent like renal tube casts. If the inflammation be severe, lymphoid cells of an inflammatory nature may be found between and under or even inside the tubes. Although the structural evidence is weak there can be little doubt that acute gastritis is a frequent affection. Thus the stomach in cardiac disease already mentioned, shows a *sub-acute inflammation*, as evidenced by the hyperæmic condition of the blood-vessels and the mucous secretion; and such a state, though less marked, may be often met with in other forms of disease, as in morbus Brightii, &c.; and in cases of Addison's disease, where great irritability of stomach has existed, some evidence of a gastritis has also been present. If we notice the symptoms attending such cases as these, we cannot do otherwise than suppose an inflammation is present. The frequency of stomach derangement in common catarrh, where the bronchi are thought to be the parts only involved, would suggest it as a not uncommon complaint; we have often found in cases of acute pneumonia and other febrile diseases, the stomach in a state of minute reddening, its surface coated with tenacious and sometimes semi-opaque mucus, different in character from the natural clear mucus of the stomach walls, and in great excess of the normal quantity; this corresponds with the loss of appetite which is experienced in such febrile states. When the microscope cannot be used, it is rather by these products of the inflammation than by any redness that we should be guided in judging of a catarrhal inflammation, for inflammatory redness of the stomach or of the skin may quite disappear after death; we would advise you always to examine the stomach, and become well acquainted with its morbid appearances.

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Croupous gastritis.—This is, perhaps, still more rare; it has been seen by Sir W. Jenner in diphtheria. Dr Delafield says, "it is almost always secondary to typhus, pyæmia, puerperal fever, cholera, dysentery, the exanthemata and irritating poisons. The false membrane is in small patches, and may line a large part of the stomach." He gives a case of idiopathic croupous gastritis, in a man of forty-six, fatal in eight days. Dr Wilson Fox has seen it in phthisis. We have seen it in a case of gouty kidney, the false membrane lining the whole organ; also in a case of abscess of the liver, along with similar croupous colitis. Otherwise, with the exception of a few unimportant patches, we have no experience of it.

Gastritis from irritant poison.— We know nothing less than an actual breach of surface, or some chemical change in the tissue, which can decide you in declaring that a stomach has been affected by an irritant, especially since the hyperæmic state found in heart disease is believed, only with some hesitation, to have passed beyond the stage of congestion. In the case of strong acids, the so-called corrosives, a chemical change occurs in the mucous membrane, and a dark brown, greenish, or black mass results; you will see how in many of these examples the poison was never carried round the stomach, or else it had become so diluted that its effects were lost; so that you see the effect only at the spot where the poison first struck the inside on entering, this being about the middle of the greater curvature, opposite the œsophagus, and thus often only the pyloric half of the organ is affected. In this case of fatal nitric acid poisoning the corrosive action has leaped from the cardiac half to the pyloric end without much hurting the intermediate part. In the stomach of a child, who died from swallowing some soap-lees, you see the œsophagus is of a brown colour throughout, and that there are one or two brown spots on the membrane; in cases of poisoning by sulphuric acid, the parts with which it comes in contact are blackened, and hardened into a thick mass; and in some cases the acid perforates the stomach. In one case two thirds of the stomach wall was completely gone, and the remainder showed no charring, or mere traces. The acid after escaping involves the neighbouring organs, which are all charred. In this case, where the woman lived several days after drinking diluted sulphuric acid, the whole mucous membrane was found lying loose within the stomach, and of a bright yellow colour. In this case of poisoning by nitric acid the interior of the stomach is of a curiously bright green mingled with dark brown and black. This one by hydrochloric acid is coal black. But, though these varieties prevail the effects of the several acids depend too much on their concentration to be quite characteristic. Caustic alkalies convert the mucous membrane to a tawny pulp; they often perforate the stomach. Carbolic acid produced a curious dry tanned-looking surface. In cases of poisoning by arsenic, the effects are not due to corrosion but to irritation, and inflammation may be slight; thus in one of the last cases occurring here, where death took place after ten hours, the inflammation was only characterised by the red colour and injection of blood-vessels, there being no ecchymosis, extravasation, nor any ulceration. In this specimen you see a patch coated with mucus containing the poison, where a circumscribed intense inflammation had occurred. In another case, that of a young man who survived six days, numerous ulcers were found. Various other conditions you will see in our preparations and drawings. In cases of arsenical poisoning, we have noticed an absence of those usual signs of decomposition the blackening and distension of the bowels, which are small, and of a pinkish colour with little gas in them, and very like the intestines in cholera. Arsenic directly prevents decomposition. Cholera sweeps away all the food that would decompose. The effects of a chronic inflammation and ulceration, leading to perforation, are seen in this specimen of a stomach affected by chloride of zinc. The woman, who was a patient of ours, drank some Burnett's solution, and survived twelve weeks. The organ is exceedingly contracted and ulcerated, and communicates by an opening with an abscess at the cardiac end. In poisoning by vegetable irritants, the morbid appearances of the mucous membrane of the stomach are very slight and fallacious, especially when it is remembered that so many of these substances act on the nervous system to produce asphyxia, or difficulty of respiration, which tends to congestion of all parts, including the stomach. Thus in a case we lately witnessed, where the mucous membrane was of an intense red colour, all the organs participated in the congestion from the mode of death. In a case of poisoning by bitter almonds, which occurred here a short time ago, the mucous membrane was of a bright pink colour from vascular injection, which must be looked upon as the first stage of an inflammatory condition; even this, however, might have been overlooked, had not the history been known.

Chronic inflammatory, and other changes in the stomach.—We just now said that the first study of morbid anatomy tended rather to the denial of such a disease as gastritis, but we have seen that in all probability it is not uncommon. In searching for the disease, we labour under a disadvantage in that it is of itself not fatal so that it is only found as a complication of more severe disorders such as phthisis, albuminuria, &c., whose symptoms often overwhelm the signs of the gastritis, and thus the anatomical changes which we see in the walls of the stomach, often cannot be distinctly linked with clinical signs of gastritis. In chronic gastritis you find the mucous membrane more opaque and thicker than natural, its colour perhaps altered to a grey or brownish tint or even a black colour, through pigmentary change in extravasated blood. The mucous membrane may be rather tougher than usual, so as to allow of its being more easily detached by pulling, but if the inflammation has been more severe the submucous tissue is thickened, white, and fibrous, so that separation is more difficult. The surface is often in that condition called *mammillated.* This you constantly see in the post-mortem room, and we have a specimen in this bottle; instead of the mucous membrane being smooth, it is covered with a number of minute projections lying close together, as if the contraction of the submucous tissue had drawn the mucous membrane into this form. This is probably the cause, for we think it is not yet satisfactorily proved that it is due to adventitious structure, or is really morbid—at least not more so than the analogous case of *cutis anserina*. If you examine sections of the inflamed stomach hardened in chromic acid, you will have no difficulty in seeing that important changes have taken place, although these do not usually amount to a disintegration of the structure. Thus you find the interstitial tissue between the tubes thickened in places; if you examine patches of such thickening you will find that the apparently new matter is made up largely of shrivelled tubes. At the same time the tubes become much less straight and parallel than they properly are, so that they look confused, and it is difficult to get sections of their whole length; their walls are thick, their contents are fatty and granular in many parts, and in some places quite disintegrated. Sometimes the mouths of the tubes are blocked up, and the deeper parts dilated into cysts. The condition of the solitary glands is not constant, sometimes they are large and ulcerated, sometimes they are wasted so as to be scarcely discoverable. We have not seen lymphoid tissue between the glands such as some describe in chronic gastritis. The whole of the changes correspond with the conditions observed in glandular organs, especially the kidney, in chronic inflammation, indeed chronic gastritis is often associated with granular kidney and similar chronic changes, just as acute gastritis is associated with acute pneumonia and other febrile disorders. Since Drs Habershon and Handfield Jones first described this condition, much attention has been given to it, especially by Dr Wilson Fox, in very able researches; our own results correspond very nearly with those of these authors, but we have been much impeded by the unfortunate difficulty in obtaining good clinical evidence of the gastritis. Often you will find much change where there were few symptoms, and little change where the symptoms led you to expect much.

The mucous membrane of the stomach may be found affected by lardaceous disease, it then looks sodden and dull, and its surface is stained of a deep walnut colour by iodine. This condition is only found as a part of a very general lardaceous disease of the viscera, and as in other parts, the disease begins in the small arteries. This state produces a liability to catarrh, as in the kidney and elsewhere.

Ulceration.—*Catarrhal.*—Ulceration, as an effect of inflammation, is not so common as might be thought; ulceration being generally cancerous, or else of that equally characteristic kind known as "chronic ulcer." It may occur, however, from the effects of poison, as we have seen, and sometimes may arise as scattered minute ulcerations in connection with acute catarrhal gastritis. Such a specimen is here, where minute ulcerations are seen on the stomach.

You occasionally but rarely meet with simple *follicular ulcers;* they are small and unimportant.

Hæmorrhagic erosion.—This is a name given to an appearance of the stomach met with chiefly in those remarkably congested organs found in heart disease, of which we have already made mention; besides the extreme congestion, the mucous membrane is found destroyed in various spots, and occupying each of these is a layer of black coagulated blood. The blood escapes by reason of a superficial destruction of the tissue at the spot, and this destruction occurs, apparently, through degenerative changes in the parenchyma. It may be that the ulcerations happen in the dying hours, and thus the minute clots in the ulcers are recent. You recognise them in the deeply congested stomach, by seeing small dark spots or streaks, which, on closer examination, are found to be coagulated blood, occupying spots where the mucous membrane is deficient. Sometimes, however, this form of ulceration occurs long before death and causes hæmatemesis. We have always found it as a part of some great congestive disease, but it is said to be sometimes idiopathic. This statement must refer to similar erosions arising from irritant poisons.

Chronic ulcer is a special form of disease, whose history has been, perhaps, more elaborately followed out than that of any other disease in the body. The mucous membrane of the stomach naturally endures the contact of the gastric juice with impunity; we shall presently mention the interesting inquiries into the causes that enable the stomach thus to resist self-digestion. It has long been thought that gastric ulcer may arise through a part of the stomach losing its resisting power, and being dissolved in the digestive fluid. Virchow gave this idea definite shape by supposing that the starting-point of the ulcer is an arrest of the circulation in the affected part. His comparison of the funnel-shaped figure of the gastric ulcer with the funnel-shaped area of distribution of a gastric artery is suggestive; and subsequent experiments, which show that ligature or embolism of the arteries of the stomach will produce ulceration, have proved that the gastric juice will dissolve not only dead pieces of the stomach wall, but also parts of it whose nutrition is much lowered. Again, the acid juice may be suspected of causing these ulcers, from the fact that they occur almost only in the stomach and in the portion of duodenum above the entrance of the alkaline bile. Yet we cannot accept this view as more than an interesting hypothesis, of partial application; for it fails entirely to explain the remarkable fact, that spontaneous ulcers are almost limited in the stomach to its lesser curvature. Again, the class of persons who are most subject to chronic ulcers of the stomach are not those in whom obstruction of the arteries occurs. We have now seen very many cases of wide-spread embolism of the different viscera in heart disease, and yet no ulceration of the stomach was present; while, on the other hand, subjects of ulcer have not generally any cause of embolism present. We have seen several cases of total obstruction of the portal vein without ulceration of the stomach. Further, the experiments of Dr Pavy and others, which show that ligature of the gastric vessels will lead to digestive solution of the part affected, do not prove that the arrest of the blood is the cause of the solution; because such ligature must so damage the nerves, lymphatics, &c., of the part as to practically kill it, or nearly kill it, and thus reduce the result to the solution of a partially necrosed tissue, which is dissolved because its peculiar vital power is gone. Dr Pavy's ingenious theory of the protection conferred by the alkaline blood, which protection he supposes to fail when the supply is stopped by ligature, is of too limited application to explain the resistance of gastric surfaces to self-digestion, for polyps and other low animals in whom there is no alkaline blood show an equal security from their own digestive fluids. Hence we believe that although it is proved that solutions of continuity in the gastric mucous membrane will arise through ligature of the arteries, yet this is not at all shown to be the cause of gastric ulcers. We are disposed to regard these as more probably due to nervous influence, and as having a close analogy with simple ulcer of the cornea; like which they affect more especially the lower orders of the people.

These ulcers appear so constantly in the lesser curvature, which you must remember is a very small part of the whole stomach, that some peculiarity of this part must be sought out to explain the association. Sir W. Gull suggested that the fixity of the lesser curvature during the movements of the stomach might create some irritation; but at present there is evidently much to be learned before we can explain satisfactorily this peculiarity of their position. However they arise, it is probable that when once the ulcer has started, digestion of its surface by gastric juice furthers its disposition to spread, and is the chief cause of that unhappy tendency to perforation which gastric ulcers exhibit; and yet how slow such digestion must be when a patient dissolves perhaps a hundredweight of meat, while his stomach ulcer is spreading through a thickness of the fifth of an inch; for some of these ulcers last many years, and are as chronic and callous as old varicose ulcers of the legs, the indolent forms of which they much resemble.

But sometimes the ulcers rapidly proceed to perforation. This is especially apt to be the case in young women, and particularly in young servant-maids. Such rapid perforation is almost always fatal, so that inspection reveals the ulcer in an early stage; its figure is then circular, or very nearly so; it looks as though a piece had been punched out, so like indeed that it is commonly called the *punched ulcer*. The opening is larger on the mucous surface, and gradually narrows toward the peritoneal surface; the edge is defined and even, and the outline abrupt and bold; usually no granulations or other signs of healing are present. This circumstance, together with the sort of people who suffer, suggests that the rapid course is due to constitutional weakness; yet this weakness is not always evinced in the general condition of the patient.

You occasionally meet with a yet earlier stage of gastric ulcer, perhaps entirely unsuspected in the fatal illness, its symptoms having been overwhelmed by those of the main disease. These we have seen as single, reniform, or circular erosions, generally near the pylorus; sometimes they are quite shallow, and a little blood extravasated in the mucous membrane around would give rise to the suspicion that hæmorrhage into the tissue, weakening it and leading to its solution, may be a cause of such ulcers. They are most often associated with Bright's disease.

When the ulcer is older and larger it has still a clean cut edge in the wall of the stomach, but its base is in a more variable state, sometimes showing decaying tissue. This base or floor of the ulcer may either be the proper coat of the stomach, or else it may be the organ adjoining the stomach at the affected part, which organ has become fastened to the stomach in the spread of the inflammatory process, and has been invaded more or less deeply by the ulcer. Thus the pancreas usually (for these ulcers are mostly at the back of the lesser curvature), or the liver, or even the spleen, is invaded, or the adhesions are to the neighbouring intestines or the anterior abdominal wall, either of which may be invaded or perforated. It is noticed that when the ulcer is on the anterior aspect of the lesser curvature there is greater liability to a perforation into the peritoneal cavity; you then may find castor oil and white mixture, &c., in the peritoneal contents; such perforation occurs in about 12 per cent. of all cases of gastric ulcer. A greater proportion of danger to life arises through hæmorrhage from invasion of arteries. Such hæmorrhage may be slight from smaller vessels, or of medium severity, gradually blanching the patient, or else it may be quickly fatal through the opening of a greater vessel, as the gastric, pancreatico-duodenal, splenic, or, in rare cases, the hepatic artery, or the portal vein.

This reminds us that *hæmatemesis* is spoken of as a distinct disease of the stomach, resulting usually from portal congestion, through heart disease, or cirrhosis of the liver. A specimen here professes to be the stomach of a man who died of hæmatemesis, but in which no ulceration could be discovered; in other instances, however, the observer has been more fortunate, and this one shows how minute an ulceration is sufficient to lay open a large artery. Dr Murchison has recorded two similar instances in which very small pore-like ulcers went directly into gastric arteries and caused fatal hæmatemesis; but sometimes the most careful search fails to discover in cases of cirrhosis of the liver the source of the bleeding, and we have to fall back on the theory of exudation, which, however, Cohnheim's experiments have now made more acceptable. In a case where a gastric vein was opened we found it varicose and the portal vein full of thrombus.

The ulcers are generally single; sometimes there are several together. These are not unfrequently found opposite each other in the stomach; this Dr Brinton explains as the effect of contact of the ulcers; but Roth, from his experiment swith lapis infernalis, concluded that common contact with some intervening irritant was the cause. The rest of the stomach is as healthy as it is found in other fatal conditions equally protracted.

Many stomach ulcers undoubtedly heal; if small, leaving very slight trace; but if larger, causing a puckered scar on the mucous surface, or even drawing in the whole of the coats so as to greatly alter the shape of the organ, causing hour-glass contraction. A very chronic ulcer may produce such contraction without healing, and so may not only deform the stomach, but cause perforation, say into the duodenum or other unusual direction. We do not think, however, that every puckered surface found at the lesser curvature implies a cured ulcer; for it may represent the disease in process of formation; we have been too much in the habit of supposing that all great structural changes have resulted from an inflammatory and ulcerative process—that where there is a new fibroid tissue there must have been an inflammation; or where a contraction, a previous ulcer. We have already shown you a thickened syphilitic larynx with an irregular mucous surface, without ulceration; and in the case of keloid in the skin the disease is always erroneously considered by the ignorant to be due to ulceration or burn: and thus we think a puckered surface, or a withering of the mucous membrane, may be due to an exudation and contraction of the material in the subtissue, without any preceding ulceration. We think it is too much to presume a former ulceration in all such cases.

We once found two bean-sized *syphilitic gummatous* nodes in the subperitoneal coat of the stomach.

Cancerous Affections of the Stomach.—We believe the different names which we give to many diseases imply merely that there are some distinctions between them, which make most of them easily recognisable; but, at the same time, these so often approach one another in character, that no very definite boundaries can be made. Thus we meet with well-marked soft medullary cancer growing in the stomach and destroying it; in another case a firmer growth, which may be called scirrhous; in another a growth which appears simply fibrous. At the pylorus the last kind may be quite local, so that it is questionable whether the name cancerous can be applied or not. Combined with these forms we frequently have a gelatinous infiltration called colloid, which, however, may occur by itself; and there is also the disease called villous cancer.

There are all degrees of activity of growth and malignancy of gastric carcinoma; and these degrees are generally indicated by the hardness or softness of the cancer. The softer the growth, the more rapid and malignant. The harder and slower forms are more common at the pylorus, so that the term *scirrhous pylorus* is currently used for cancer of that part; although you do sometimes, as in this instance, meet with soft cancer there. Sometimes the disease is of very slow growth, and on examination the appearance is of a thickening about the pyloric orifice, as though from some hypertrophy of the muscular coat and submucous tissue. This preparation came from a man who died of renal disease, but had some vomiting, and the pylorus was found thus thickened. We can scarcely indeed apply the term cancerous to it, nor even to this more marked example, where the disease existed four years, and caused the death of the patient; for you see it is quite local, and does not at all invade any of the surrounding tissues; moreover, the new material thrown out is very hard and fibrous, having no juice, as cancer should have. The section shows very well the appearance of a scirrhous pylorus; before it was opened, the enlargement of that part of the stomach produced a tumour of the size of a hen's egg, and it was moved by its weight considerably lower than its usual site in the abdomen.

In such cases it would be fair to question the cancerous nature of the growth, and even microscopic examination may show so large a proportion of fibrous tissue that a doubt may remain. Here perhaps more than at any other part of the body do we see gradations between simple fibrous tissue and cancer.

But a typical *hard carcinoma* of the stomach is less ambiguous in its characters. It appears as a solid hard thickening of the coat of the stomach, generally near the pylorus, tending to surround this, not passing into the duodenum, but extending towards the cardia, especially along the lesser curvature, and having generally a well-defined raised border. Section shows the thickness—about one half to three quarters of an inch in all—to be made up of the coats of the stomach in an altered state, but still recognisable. The muscular coat is best seen with its greyish pellucid bundles strongly marked, and separated by white septa; this forms nearly all the outer layer, and composes about one third of the whole thickness. The rest of the bulk, about two thirds of all the thickness of the mass, is composed of cancerous submucous tissue. The muscular coat may be quite transformed to cancer in the more central parts of the growth, or the new substance may only separate the muscular bundles; in either way it reaches the subperitoneal tissue, puckering and nodulating the peritoneum; or causing a subinflammatory adhesion to adjoining organs, and finally an invasion of them. The glandular mucous membrane itself is often raised over the tumour, and generally soon ulcerated more or less; an excavation forming with a thick raised wall, and a more or less sloughing base.

The *medullary* form of carcinoma affects rather the cardiac orifice of the stomach (there is a notable tendency of cancer to affect the orifices; in this it differs from ulcer). The medullary kind of cancer has a more active transforming power, so that you will find the muscular coat altogether disappearing in parts; the cancer reaching the peritoneal tissue, and raising this in larger or smaller nodules. Medullary carcinoma, however, spreads with chief violence in the softer submucous layer, so that this is disproportionately enlarged, and may grow into great lobular masses, which are very liable to suffer from either deep ulceration or digestive action, so as to slough away in patches; giving then rise perhaps to extensive bleeding sores, or even opening large arteries, as the splenic; but except through such sloughing the bleeding in cancer is slight. This sloughing away of cancerous masses may perhaps reopen the food channel previously obstructed by their growth, and so bring great relief and comfort to the patient in this miserable disease. But the sloughing may extend through the whole wall of the organ, so producing fatal peritonitis.

It is impossible to describe the horrible appearance which the foul, ragged, sloughing masses in the interior of such a stomach sometimes display.

If a line be drawn from one inch to the left of the œsophagus to a point on the lower border of the stomach four inches from the pylorus, the part to the left of this line, including rather more than the great cul-de-sac of the stomach, will be found to suffer very rarely from cancer. The rest of the surface, the right and upper part, is the peculiar seat of cancer; hence it is to the organs in contact with this part that the disease is apt to spread by contiguity. Thus, the liver may be invaded so that several square inches of its altered substance lie exposed in the stomach in the shape of a sloughing cancerous sore. The pancreas, or pre-vertebral tissue, even the vertebræ, may be attacked, or the colon, or the duodenum, or rarely other portions of the small intestine, may be opened. Such openings from the stomach into the intestine are more frequently caused by cancer than by simple ulcer. The lymphatic glands share in the cancer, and by them the biliary duct, the vena cava, or the vena portæ, may be compressed or invaded. We have seen the gastric and portal veins stuffed with such cancer. Besides this local extension to contiguous organs, secondary cancer is apt to appear in the form of round nodules in the liver or lungs, or on the mucous membrane of the small intestine, &c.; in fact, in the usual seats of secondary cancer, with especial preference for the liver. The gastric cancer itself is primary in nearly all cases; one example of secondary cancer, found by Cohnheim, in the form of circular patches in the mucous membrane of the stomach, being almost unique; but such secondary patches are far from unfrequent in the small intestine.

The effect on the peritoneum is to cause a slight local peritonitis, or in rare cases, of which we have seen two, a general peritonitis may be set up without perforation of the stomach by the cancer.

As to the organ itself, the remainder of its mucous surface is generally healthy. If the cancer obstruct the cardiac orifice the stomach is empty and small; if the pylorus, it is large, often very large indeed. If there is a considerable cancer of the pylorus its weight may drag that part down, so that it is found much below its natural position. This fact you must remember at the bedside, for the pylorus may thus descend even to the brim of the pelvis.

Twice we have known a chain of cancerous glands to extend from about a cancerous stomach, and to appear as cancerous nodules in the root of the neck.

Villous cancer.—The cancer sometimes implicates especially, and takes its character from the most superficial layer of the gastric mucous membrane, showing then a tendency to form processes which may grow and branch very freely, producing a richly clustered mass of dendritic vegetation, subject, however, to defacement by sloughing and ulceration. The structure of such processes is often very beautiful. Each is composed of a central blood-vessel clothed with well-formed "cancer-cells." The malignancy of some examples of this cancer was extreme, so that the growth invaded the liver by contiguity, a great mass of soft carcinoma spreading into it from the floor of the papillose ulcer. We shall presently speak of superficial villosities which have no such cancerous nature. (*Fibrous papilloma*, p. 392.)

Epithelioma.—We have seen a few cancers of the stomach which showed, microscopically, the most perfect glandular structure, resembling, that is, the mucous glands of the intestinal wall. Such cancers look like medullary carcinoma; we shall describe them more particularly in their favourite seat in the large intestine. Secondary to such cancers we have seen similar glandular structure in the liver.

Colloid.—We have already mentioned this disease as especially affecting the peritoneum, and in the most marked cases we find the whole of that membrane, including the serous coat of the stomach, occupied by the deposit. Colloid cancer of the stomach is generally a compound disease; for example, a scirrhous cancer is associated with colloid, and when a section is made the new matter is found to be softer than ordinary scirrhus; on closer examination a gelatinous substance is seen mixed with it, and may be picked out with the point of a scalpel. In these cases the muscular tissue appears more destroyed than in carcinoma; the peritoneum is invaded too, and thus the disease is a more uniform infiltration of the stomach wall; also it spreads generally more widely. This specimen of a section through the pylorus shows the general infiltration of a gelatiniform matter. A purer and simpler form of colloid is seen in another case, where no ordinary cancer appeared in any part of the body; the whole of the coats are infiltrated with the jelly-like matter, the mucous membrane is raised, and on looking at its surface, it appears translucent from the pouring out of that substance beneath it. This one is a remarkable example of colloid affecting the œsophageal end only. Colloid has little tendency to ulcerate, or to form secondary nodules in the liver, but is apt to light up general colloid disease of the peritoneum.

Sarcoma.—Occasionally, without any disposition to ulcerate, a growth may extend slowly within the gastric coats, and maintain its firm character, as though simply fibrous; but instead of consisting of simple fibre tissue, it is seen to be composed of nucleated fibres, like those which compose *recurrent* or *semi-malignant* growths, and which are known as sarcoma; such growths as are frequent in the external parts of the body. In these specimens you see the organ immensely thickened throughout more than half its extent by this adventitious deposit. In one case the pyloric half is thus affected, and in another the half comprising the lesser curvature.

In some instances there is black pigment in such sarcomatous growths forming *melano-sarcoma*, or, shortly, melanosis of the stomach. It may be secondary to melanosis of the eye or skin: it is very rare.

Myoma is sometimes met with, usually as a hard, white prominent mass, into which the muscular coat expands; sometimes such a tumour projects considerably, forming a *polypus*. In this specimen you see one or two small growths consisting of fibro-cellular tissue, with an admixture of muscular fibre, and here are three such pedunculated tumours found accidentally in the stomach of an old woman. Their structure would entitle them to the name of myoma or *myofibroma*, and they correspond to the descriptions of this condition which are published. Cases are recorded where a polypus of this kind has been vomited up.

Fibrous papilloma.—You meet with specimens of villous growth of a very simple kind springing from the gastric surface. The microscope shows these villi to resemble very much the similar processes of the chorion of the ovum, and when the two are placed under water the resemblance is very exact. When of this simple kind they spring immediately from the mucous membrane; we shall have to show you the same in the bladder; they have only the slightest fibrous basis. These growths are now described as *fibroma papillare*, and must be distinguished from villous cancer.

Small *lipomata* occasionally occur in the submucous tissue of the stomach.

Adenoma, in the form of local hypertrophy of the gastric glands, is described. The occurrence of *lymphoid* tissue in the stomach in leukæmia we shall mention again when speaking of that disease.

Cysts and pouches.—We showed you a cyst in the œsophagus which we lately met with in the post-mortem room, and we have seen a very similar cyst of about the size of a walnut in the walls of the stomach. Here is a specimen of a pouch, which may have been originally a closed sac, but is now broken into; and if you look on the museum shelves you will find a similar preparation dried. Cysts of small size from dilatation of the glands are not very unfrequent.

Cadaveric Softening.—As in describing many of the morbid appearances of the stomach we have referred to post-mortem changes, it will be convenient to speak next on this subject; and we must again impress you with the importance of studying these changes, since they are much more striking than many appearances which may be due to disease. You know that the gastric juice after death acts on the coats of the stomach, to dissolve them in the same way as it does ordinary flesh; it has generally been thought that the juice retains its strength, while the stomach itself loses its vitality, and thus becomes subject to the solvent influence of its own secretion. Bernard, however, has shown (and you have seen Dr Pavy repeat the experiments) that the solvent power of the gastric juice is equally great on living tissues, hence it is thought that the reason why the stomach is not affected during life, is due to the constant evolution of mucus and epithelium, which protect it; or, as Dr Pavy believes, the alkaline blood in the coats prevents the acid juice from attacking them.

Such explanations are not, however, of sufficient breadth, for they do not explain why the powerful juices of the fresh-water polyp, for instance, which will dissolve a water-flea through its insoluble coat, will not dissolve the bag-like body of the polyp himself. And we think that the reason of the stomach's self-protecting power must be set down to unknown vital properties of the gastric membrane, similar to those which in a viper's poison-bag prevent the viper from being infected with his own venom. However this may be, the gastric juice after death will dissolve the stomach; but the juice appears to be secreted only by the stimulus of food, so that if the patient die with the stomach empty, it may be found several days after death unaltered, with its mucous membrane entire, and its rugæ prominent and unchanged.

If, however, there be food in the organ, and the digestive process going on, then the mucous membrane will be found to have undergone solution, and, in some very rare cases, all the coats will be found to be destroyed, leading to perforation. The extent of stomach affected corresponds to the quantity of food within it. The part affected depends upon the position of the body after death. As this is generally supine, it is the larger end of the stomach, at its posterior part, which is usually acted upon; and if the contents be removed, you can see by the change which has occurred to what height the food reached, the water-mark being accurately defined by a distinct margin; above this margin, in the pyloric end of stomach, which lies superior, the natural rugæ of the membrane are seen. In the most depending part which has held the food, what is mostly seen is a softening and thinning of it, so that it looks like a layer of clear mucus, or there is a complete loss of the mucous membrane, when the submucous tissue is exposed; or this may be also removed and the muscular coat softened and thinned, or even the peritoneum perforated. The affected part differs from the rest of the organ, not only in thinness of its coats, but in being smooth from the loss of the rugæ; it also presents an appearance, from the action of the gastric juice on the blood and vessels, which is very characteristic and is resembled by no pathological state. You know that the gastric acid renders the blood black, and thus, when the vascular submucous tissue is exposed, large vessels are seen coursing along in it of a black colour; but the blood has exuded through their walls, and thus you see an irregular arborescent appearance of ill-defined black lines running over the dissolved part. This you here see in a preparation and a drawing; the latter showing admirably the process of digestive solution. In cases of perforation from digestion of all the coats, the contents of the stomach are found in the abdomen; we have already alluded to such a circumstance occurring in the œsophagus, with escape of the gastric contents into the chest; and occasionally the duodenum may equally suffer. Such a perforation is easily distinguished from a perforation through disease both by its position and character; its position at the cardiac end is unusual for ulcer; and again, an ulcer arising in an inflammatory process, its edges will be found thickened from lymph, fibroid tissue, &c.; whereas in case of softening the opening is large, the edges thin, dissolved, flocculent, ragged, and hanging in shreds. The opening is much less abrupt than that found when a corrosive poison has perforated the stomach.

It is not clear what are all the circumstances necessary for the production of this extreme solution. There can be no doubt that a healthy digestive process in operation just before death is the most favorable for it, and thus it is that animals killed with food in their stomachs are so often found to have the organ dissolved; and for the same reason it is in persons who have died suddenly during digestion that the stomach is most affected. Constantly, however, as we are meeting with persons dying under these circumstances, it has not been especially in them that *perforation* has been met with, for during last year the only two cases observed were one in a child who died of brain disease, and another in a lad, with albuminuria; in another more recent case there was disease of the pons varolii. It has been thought that the ordinary processes of decomposition have had their share in its production; but this does not seem so, for now and then it falls to our lot to examine bodies frightfully decomposed, but in none such have we found perforation. It has been thought, too, that warm weather has influenced it; but this notion had arisen probably from the supposition that decomposition is more rapid in warm weather than cold, which is by no means the case; the condition of atmosphere which most favours decomposition is moistness, and thus a moist cold day in winter is more favorable to the general disintegration of the body, than a warm dry day in summer. This we have observed on repeated occasions. It is clear that warmth would favour solution, but then it is manifest some other cause must be in operation to determine it in one case more than another; and it may be due, perhaps, to the formation of a morbid excess of gastric juice; this, however, has yet to be proved.

Idiopathic Softening.—We mention this in connection with post-mortem solution, because it is stated by some writers to be a frequent pathological condition in children. It is called *gelatiniform softening* of the stomach, and described by some Continental authors as productive of certain symptoms, and leading to a fatal issue. Perhaps it is not right to doubt any facts well observed by those whose experience in children's hospitals gives them so favorable an opportunity for making such researches; but we must confess our doubt about the condition described. The examination of cases where vomiting and other symptoms denoting the kind of gastric disturbance associated with this softening were present, showed in the stomach no greater degree of softening than would be accounted for by the natural softness of a child's tissues, combined with a post-mortem digestive process.

Morbid Contents.—You should always examine the contents of the stomach after death, to see whether they consist of food or merely mucus, and to observe their colour; if brown or black, it may be owing to blood or some medicine administered; blood may come from the nose, chest, &c., or from a fractured skull. Chemical tests will detect iron, as the microscope will blood.

The *quantity* present is sometimes significant. Thus we have found two pints of food in the paralytically dilated stomach of a fever case.

Some think that you may find cancerous growth in vomited matters. This is just possible, but the most interesting object met with is one of modern discovery—the *sarcina ventriculi*, a vegetable fungus discovered by Goodsir. The sarcinæ are at once recognised in the contents of the stomach by their cubical figure, each face being made up of four small cubes, and thus resembling a pack with a cord round it, whence the name sarcina. Many of these may be united together, forming large bundles, and occasionally they have a greenish cast: they are found in those cases, especially of obstruction, where the food ferments.

We must notice the occasional occurrence of parasites which appear to correspond to the *bots* with which the stomachs of cattle are affected, and which proceed from a fly called the *Œstrus bovis.* A certain doubt must attach to all kinds of maggots and other wonders shown you by patients; but some of the cases are so well authenticated that there can be little doubt that the œstrus does, very occasionally, affect man.

Air.—You not uncommonly find gas in the form of bubbles beneath the mucous membrane. In all such cases which we have seen it has arisen from decomposition: a true emphysema from extravasation of air we have never witnessed.

INTESTINE

Malformation.—This shows itself chiefly in two places. First, at the seat of the fœtal vitelline duct, which originally passes through the fœtal umbilicus to join the umbilical vesicle. The persistence of this duct appears to give rise to a diverticulum from the ileum, which you will frequently see in the post-mortem room, and of which there are specimens here; such diverticula generally jut from the lower end of the ileum, about two or three feet or more above the cæcum; nearly always on the free border of the bowel. They are of various lengths; you may find them as pouches ranging from the smallest size to portions several inches long, like the cæcal tubes in birds. Their structure is exactly like that of the neighbouring part of the ileum, and they partake in its morbid processes, so that, as in Dr Galton's case, typhoid ulcers may be found in them. The diverticulum may extend through the umbilicus of the fœtus, and so be divided when the cord is cut; it may then be attached or not to the umbilical opening. Under these circumstances a plastic operation may close the bowel, but afterwards strangulation by the band thus formed has been known to occur.

The second principal seat of malformation is the end of the bowel. The simplest and most important condition of the kind here is absence of the lower end of the rectum—*atresia ani*—requiring operation to establish an anus. A greater length of colon may be wanting, so that to reach the gut, operation in the loin or groin is required. In much rarer instances the small intestine may be blind at one or many points; such badly malformed cases are, fortunately, unviable; as, also, fortunately, are most of those conditions where a cloaca is formed by non-development of the divisions between the genital, urinary, and fæcal passages. In these, various combinations of the rectum, vagina, and ectopic bladder are met with. In cases of yet less complete development the ileum may open upon the ectopic bladder.

Malposition of the intestines, either as herniæ or as displacements within the abdominal cavity, are common. When the liver is flabby it sometimes falls back, and the colon rolls over its edge, separating it from the anterior abdominal wall, making the liver appear much smaller to percussion than it really is. Internal displacements may arise through adhesions of the intestines in early life to other parts, so that they are prevented taking their natural places during the growth of the body; when the position is, as in many cases, greatly altered, it is tolerably certain that the union must have occurred in fœtal life; and this becomes another illustration of what we told you, when speaking of malformation of the heart, that such conditions often arise from disease in utero. In the present instance we can speak with more certainty of the occurrence of such inflammation, for we have several times met with peritonitis, both recent and old, in the new-born child, especially in syphilitic infants. Adhesions occurring in extra-uterine life, no doubt, alter the position of parts to a certain extent; and thus we find the colon drawn down to the pelvis, or the sigmoid flexure carried upwards, &c. Such displaced parts may twist on themselves; or else form loops, under which coils of intestine may become strangulated.

But twists of the bowel often occur without any preceding adhesions. Such a twist, however arising, is called *volvulus*. When this accident has come under our observation, there has evidently been unnatural length of the mesocolon. It usually occurs in elderly males, whose time of life would show that the lengthening of the mesocolon is an acquired change. Volvulus happens most frequently at the cæcum or sigmoid flexure, but it has occurred in other parts of the colon, and even in the small intestine. Sometimes the canal twists on its axis; sometimes a coil twists on its root. A coil so twisted may dilate to an immense size, while it becomes very dark purple in colour. Thus a twisted sigmoid reached the under surface of the diaphragm. Sometimes the twist is more complex—one coil is wrapped around another coil; when this is so, there is generally some awkward adhesion of the coils together causing the twist. We have already mentioned the curious twists to which the bowel is subjected by contraction of peritoneal cancer. This may cause fatal ileus, generally very gradual in its approach, and perhaps hidden amongst other symptoms.

Hernia.—Another kind of malposition forms the great and important class of intestinal hernias, where the bowel escapes from the abdominal cavity. The more ordinary kinds of hernia, namely, *inguinal*, *femoral*, and *umbilical* are so fully treated of by the surgeon that we shall not dwell upon them, but will mention to you only those rarer kinds which are usually discovered at post-mortem examinations as causes of abdominal obstruction obscure during life. But this reminds us that before dismissing femoral hernia we must earnestly warn you against overlooking the slight swelling which may be all that is offered to you as sign of a hernia into the femoral canal; such a hernia may prove fatal while the patient denies having had any pain in that part, and when the swelling is very doubtful. We will also get you to observe that when the bowel in hernia sloughs open, the breach is along the line of stricture; but if it be burst in taxis the rent may be in the prominent part; such accidents may produce fæcal abscess. Among the rarer places where hernia forms is the *obturator* foramen. Here, again, it may occur without any pain or tumour. Another is the *ischiadic notch* in the course of the sciatic nerve; this is a very rare occurrence. A hernia rarely forms behind and beside the anus, through the hinder part of the perinæum, *perinæal* hernia; or into the labia pudendæ, *pudendal* hernia.

Ventral hernia is like umbilical, but passes through some casual weak spot which allows an opening in the fascial coats of the abdomen, in the middle line or to one side of it. Besides these forms of hernia the intestine may force its way outwards by bulging in the wall of the vagina, and protruding from the vulva, *vaginal* hernia; or in a similar way through the rectum, *rectal* hernia, protruding through the anus.

Diaphragmatic hernia is generally caused by grave injuries bursting the diaphragm, so that the patient is killed quickly by the accident; but cases are recorded wherein the patient has survived such an accident for a long while. There may be no gastric symptoms, as in a case recently under our care. Other examples are formed more slowly through yielding of the weak portions of the diaphragm, and some are congenital; these are, however, rare. Another interesting kind of hernia is known as *retro-peritoneal;* it is very unfrequent. The intestine pushes down behind the left mesocolon, passing in above, and down behind, the inferior mesenteric artery, into the lax subperitoneal tissue; or, in one case, behind the superior mesenteric. In this way a large downward pouch may hold nearly all the small intestine. We have once seen an upwardly directed pouch behind the right mesocolon turning in below the mesenteric artery. We will remind you that a hernial sac may be returned into a subperitoneal position with the stricture unrelieved.

Incarceration.—This condition, allied in its results to the last, is not a malposition, but rather a misfortune of position in which the bowel becomes imprisoned and strangulated by a band around it; this is a relatively frequent cause of death. Such a band is generally either a diverticulum of the ileum, or else the cæcal appendix. The latter, as you know, is excessively variable in its position, sometimes being altogether behind the peritoneum, and sometimes free in its whole length. We have known it to form the only contents of a femoral hernia, and we have found it touching the gall-bladder; but it may be attached at its end and free in its middle, so as to form a loop always fishing, as it were, for an unhappy coil of small intestine. A diverticulum of the ileum attached at its end becomes a similar snare. In some cases this diverticulum appears as a solid cord, representing, as it were, the vessels that once accompanied the vitelline duct, but sometimes such cords are too high for the vitelline duct. Besides these, the more usual causes of external constriction, we have seen the colon crossed at its splenic flexure by a simple thickening of the peritoneum and so strangulated; or there was a hole in the omentum, a mere gap, looking the most casual and innocent thing, through which only one knuckle of ileum had got; or in the mesentery a similar hole through, or a pouch formed within it (such a pouch constitutes one sort of extra-peritoneal hernia); or the omentum attached to an ovarian tumour, or to an extra-uterine fœtation, may cross the intestine; or so may an adhesion caused by tubercular ulceration within the bowel; or a loop produced by adhesion about the appendages of the broad ligament of the uterus; any such cause of external constriction may catch the bowel, especially the small intestine, and cause perhaps slower obstruction, but far more generally a quick and fatal form of ileus.

Intussusception.—This disease, judging at least from our own experience, is not nearly so common in adults as is generally supposed; and as a cause of fatal obstruction in the bowel, is by far less frequent than the bands of peritoneal adhesions already mentioned. Before morbid anatomy was as much cultivated as at present, it was commonly assumed in cases of internal strangulation, where symptoms were sudden and speedily fatal, that such an introversion of the intestine must have occurred; we should say, however, that rapidity of symptoms rather militates against the probability of intussusception, seeing that the process of its formation is mostly slow and the impediment at first partial. We have seen but few cases of intussusception in the adult, and it is remarkable that these have frequently been occasioned by growths in the wall of the bowel, especially in the colon. In one of our cases of intussusception of the ileum, in a girl of seventeen, the starting point was at a diverticulum of the ileum. Another in the ileum, examined by Dr Cayley, began in cancer there, a rare disease, indeed, in that situation. In these cases the obstruction is generally never complete, and death does not occur for some weeks. In infants, however, the affection may rapidly develop, and be speedily fatal. It is only where the introversion is considerable that symptoms occur; it is highly probable that a slight slipping in of one portion of bowel into another is frequently taking place, if we judge from what is met with on the post-mortem table; in children especially, who have died from cerebral disease, the intestines are found contracted, and often invaginated in several places. These are readily pulled out. The occurrence in this class of cases naturally suggests a nervous influence productive of the condition; it is mostly found in the small intestine. The graver intussusceptions are also more frequent in the small intestine. Thus, among twenty-nine cases of separation of the invaginated part collected by Dr Peacock, the piece of bowel was found to be ileum in twenty-two examples. A common seat also is at the end of the ileum, which enters the cæcum.

The part that first enters continues to keep the most advanced position of the whole intruded mass in its further progress. You can easily see how the passage is to a certain extent pervious, by attempting to imitate the invagination on the finger of a glove: if you simply force one part into another, and then cut off the top so as to leave the channel open, it will have some resemblance to an intussusception. Students are often at the moment puzzled, when asked to enumerate the layers met with in a section of an intestine so invaginated; but it only requires a moment's consideration to know their relations.

There are three portions of tube including and included. The inside one joining the middle at the most advanced or deepest part of the invagination; the middle one joining the external at the least advanced part; between the inside tube and the middle one is a recess of peritoneal surface in the direction of the course of the intussusception; this recess may contain some blood and lymph; between the middle tube and the outside one is a recess of the mucous surface closed in the reverse direction. If now you examine these three tubes of bowel you will find that they suffer in very different degrees and always quite characteristically; the part that suffers least is the outermost tube, while the part that suffers most is the middle tube, especially at its junction with the innermost. This junction is the original starting-point of the intussusception, and if a cancerous tumour caused it, it is here you find that tumour. If there is no cancer you might often be led to think one present, through the surprising swelling of the bowel wall at this bend. We have measured the bowel three quarters of an inch thick here, its mucous surface is intensely congested and eroded, or hangs in sloughy shreds, hence the bleeding which is so characteristic of invagination of the intestine.

To see the amount of mischief thus produced you must open the containing bowel, or sheath, and thus expose the included portion within; you then discover that the example of the invaginated finger of the glove does not perfectly hold good, nor, indeed, the ideal or diagramatic drawings of an intussusception, for in these the tube is supposed to be simple and free, but the intestine has a mesentery attached, which acts a very important part in the process; it is this membrane which prevents intussusception from more frequently occurring, but if once the intussusception has taken place, the mesentery attached to one border of the bowel pulls on it so as to make the mouth of the enclosed part turn to the side to which the mesentery is attached, and presses it against the side so as almost to close the orifice. Thus, when you open the outer intestine, or sheath, you do not find the opening of the bowel at the extreme end of the intensely congested mass within, but turned to one side of it. This dragging of course obstructs the vessels of the mesentery, and, consequently, this structure is found of a dark colour, and sometimes indurated from blood effused within it. The seat of stricture is always at the junction of the middle with the outer tube, and all the included part is strangulated; it becomes of a dark colour and, perhaps, gangrenous, so that it tends to become separated; and during this process the opposed serous surfaces of the inner and middle tubes, at the point where the one portion of bowel enters into the other, are becoming united by lymph which is formed through inflammation. This is the process of cure; but during the sphacelation and separation death often occurs, through an ulceration of the intestine at the point of junction, or an extension of the inflammation through to the peritoneum. Recovery is sometimes complete; this is, however, rare. The patient generally suffers afterwards with signs of abdominal obstruction. Thus, we have met with two instances of fatal annular stricture of the ileum with puckering of the mesentery at the spot affected, all no doubt due to former intussusception, though no history of this was obtained; others have recorded similar cases. In explaining the absence of history you must note that the piece of bowel passed may be small, and recovery is often then remarkably rapid. The piece of bowel passed varies from two to forty inches in length; but in one remarkable case, quoted by Dr Peacock, eight pieces, passed in three years, amounted in all to no less than twelve feet.

Other causes of obstruction.—We may here mention the other causes of intestinal obstruction. Among them are those seated in the wall of the bowel. Thus simple, tuberculous, syphilitic, or cancerous ulceration may give rise to *contraction* of the bowel, or the cicatrix left by former ulcers, or by the separation of an intussusception, may for years produce chronic intestinal misery ending in ileus. Stricture of the rectum may be produced also by implication of the gut in pelvic abscesses connected with the internal genitalia; such abscesses may open into the rectum, and by contraction may twist or bend it so as to cause chronic obstruction.

Prolapsus ani is a condition resembling intussusception, only that the external tube is awanting. If the prolapsus is small, it is of no great importance; but if great, a strangulation of it similar to that which causes gangrene of an invaginated bowel may occur, and although gangrene is rare, yet the extruded part may become congested and inflamed, swelling to a large size. The prolapsus of congested mucous membrane only, may be mistaken for a prolapsus of the whole bowel.

Lastly, yet another kind of obstructive agency is found in *concretions* stopping up the canal; usually large gall-stones, which have made their way into the duodenum by ulceration from the adherent gall-bladder, or which may even have passed down the dilated bile-duct. These may be stopped in the narrow portion of the ileum and cause fatal ileus—we have here several such preparations; or else intestinal concretions may act in the same way, such concretions being composed of hair or woody fibre, magnesia, &c. In some rare cases ileus is produced by enormous accumulations of fæces in the lower end of the colon. But the danger from these accumulations is rather that they tend to produce ulceration, and perhaps perforation, of the bowel.

Dilatation.—Enlargement and distension of the intestine, especially of the colon, is found above the seat of chronic stricture, sometimes reaching an enormous extent. The muscular coat at the same time is hypertrophied, so as to be twice to four times as thick as natural. The effects of stricture on the dilated intestine may be very severe, especially if purges be given. Thus, we have sometimes seen the peritoneal coat and the muscular coat cracked in several places, when the colon has contained eight pounds of fluid above a stricture at the sigmoid. This giving way occurred at the cæcum, and not near the sigmoid. Also we have seen the whole thickness sloughed through over several square inches; or the mucous surface may be ulcerated more or less severely; this also may happen at some distance above the stricture. We have met with some cases in which the intestines were punctured when distended with gas, and no ill result followed; but twice peritonitis occurred in similar cases.

Sometimes you meet with cæcal pouches from the colon. These are hernial protrusions of the mucous membrane outwards through the muscular coats. They appear to be innocent; sometimes they are numerous; they occasionally contain fæcal matter, and might undergo ulceration and perforation, but we do not know of this as having occurred.

Injuries.—These are mostly fatal, from the fact of some of the intestinal contents being extravasated into the peritoneum, and setting up inflammation; otherwise wounds of the bowel may be healed. Thus, if the wounded intestine should protrude, or can be secured and stitched up, a cure may follow, the threads finding their way ultimately into the canal and being so discharged. Several such cases are recorded. Generally the mucous membrane protrudes at a wounded spot, through retraction of the muscles; but we have seen the reverse occur, the seat of puncture dipping in towards the channel of the bowel, and the peritoneal surfaces around the puncture being thus brought in contact, so as to cohere and favour a cure; not, however, enough to prevent fatal peritonitis. Many of the injuries to the intestines which we witness occur from falls, or from wheels of vehicles passing over the abdomen. The parts most exposed in run-over cases are the duodenum and jejunum; for the ileum, in the lower part of the abdomen, is shielded by the pelvic bones, while the lumbar spine rising forwards, without ribs or pelvis to take the pressure, offers a hard surface upon which any unfortunate piece of bowel may be crushed by the wheel. Often also the intestine is ruptured by the kick of a horse, or by any other sharp blow, or a fall; and it is very important to remember that such a fatal injury may occur *without any external bruise.* The intestine may be entirely torn across; the mucous membrane then protrudes, while the peritoneum is simply divided, and the muscle retracts. Sometimes the bowel is bruised and inflamed without bursting, and peritonitis is set up.

Congestion.—This is generally of a passive kind, and associated with a similar condition of the stomach, as already mentioned, among the consequences of heart disease, &c., engorgement of the portal system occurs, and thus congestion of the mucous membrane of the stomach and intestines. The congestion is, however, more direct in primary hepatic obstruction, as cirrhosis, &c. Owing to the resistance to the current in the portal vein, the serum escapes on the peritoneal surface, constituting ascites; while on the mucous surface the blood escapes entire, and so may be found in the intestine. If there is no bleeding, the mucous membrane is highly congested. We have just now alluded to mechanical impediment to the flow of blood as producing bleeding in intussusception of the bowel where the congestion is excessive. You must not be misled by congestion from mere accidental dependence of the coils. The blood settling down to the deepest part of the coils produces an unequal congestion that surprises beginners. We have seen great congestion in pyæmia, and this has suggested a cause in a morbid state of the blood itself. You know that in septicæmia produced by experimental injections of putrilage into dogs' veins, the chief symptom is diarrhœa. When in slight degree, congestion may perhaps arise from an irritant in the shape of food or medicine, the redness is then observed more on the rugæ; if there be any large quantity of mucus or lymph adherent to the prominent parts, we must consider that the hyperæmia is inflammatory.

Œdema.—In cases of dropsy you sometimes find the whole structure of the bowel in an extreme condition of œdema, so that its weight is very much increased, and it has a gelatinous appearance. This condition must greatly impede the functions of the bowel, but we have not been able to trace any special symptoms to it. It appears sometimes to be increased by irritants, as croton oil. We have thought that it sometimes had obstructed the mouth of the bile-duct, causing jaundice.

In persons who die during digestion of a full meal, you may see the villi whitened by chylous fluid within, also the lacteals traced out plainly as white lines.

Lardaceous Disease.—This is rather frequent in the alimentary canal; it produces a state of the mucous membrane more easily recognised than described. The surface looks like wet wash-leather; if it be cleaned and iodine applied, it becomes of a deep walnut colour. Microscopic examination shows the minute arteries chiefly affected; but the amyloid matter extends from these into the tissue around. Peyer's patches generally are less implicated, which is remarkable when you remember their structural identity with the Malpighian bodies of the spleen, for these are always first affected in that organ. If you are searching for lardaceous disease, choose a Peyer's patch and the part around for application of the iodine, and the contrast of effect will guide you. Death by diarrhœa is common in these cases, for there is liability to catarrh, or even severe enteritis, which may lead to sloughing.

Inflammation of the Intestines.—The term enteritis, or inflammation of the small intestine, has generally been used to denote certain symptoms of derangement of the bowels, with a more vague signification than would be given it by the morbid anatomist.

General enteritis.—A general acute inflammation of the whole interior of the intestinal canal, without any obvious reason, as a special affection to which we can give the name idiopathic enteritis, is certainly not common, if it exist at all. At least, we have never seen it.

A general acute catarrhal irritation of the alimentary canal occurs under the same conditions as the quite similar catarrh of the stomach, *e.g.* in pneumonia, fevers, croup, &c.; it causes a pinkness or redness of the mucous surface, and an increase in the quantity of mucus on the surface, which mucus becomes also semiopaque.

A chronic catarrhal inflammation is shown by enlargement of the glands, and by darkening of the surface to a dusky, slaty, or even to a black colour (this black colour disappears in spirit-kept preparations). There is an excess of mucus on the surface. In some states of chronic irritation, the solitary and agminated follicles become blackened, producing an appearance well compared to that of a shaved beard. Often in the post-mortem room we cannot connect these appearances with definite symptoms, but there is no doubt that they belong to old chronic *enteritis* of a degree milder than would attract remark among the symptoms of the fatal illness. They most frequently occur in cirrhosis of the liver, cardiac disease, or other causes of portal congestion.

General enteritis, distinguished as "toxic," may be set up by irritant poisons, and therefore in suspicious cases the whole canal should be carefully examined. You will usually find more severe effects in the large than in the small intestine. And, indeed, it is remarkable how the poison may pass down the small intestine without affecting it, and then set up violent inflammation in the large. This has especially been the case in two or three instances of poisoning by bichloride of mercury which we have seen, where the small intestine had escaped with impunity, but the large had even had its mucous membrane destroyed. On the other hand, in a case of phosphorus poisoning, the small intestine was more affected than the colon.

Local enteritis.—The long tract of the intestine is liable to be affected with many changes that are included under the wide term inflammation; also in the variously constructed parts of the canal some inflammations elect one seat and some another; so that local names are given to the inflammations of special seats, such as, *duodenitis*, *ileitis*, *colitis*, *typhlitis*, &c. Besides these varieties as to the *position* of simple inflammations, there are special *kinds* of inflammation, such as *tubercular*, *typhoid*, *dysenteric*, &c., but of these we shall speak afterwards.

If the duodenum is especially affected, the disease is called *duodenitis*. The changes which no doubt are constantly arising in the stomach from improper food are propagated to the duodenum, and it is even thought that some forms of dyspepsia are due to its inflammation; and that a continuation of such affections up the bile-duct produces jaundice and other affections of the liver. The duodenum may certainly be found highly congested in the same case where the stomach suffers. In poisoning, too, the duodenum shares the inflammation. Ulcers are met with in this part of the intestine relatively often when they exist nowhere else. On many occasions we have met with these. These ulcers correspond in their general character with gastric ulcer (p. 385), and like these are often associated with Bright's disease. They sometimes give rise to perforation and fatal peritonitis. As to their cause little is known; but these ulcers occur in the upper part of the duodenum, and this part is in contact with acid contents, like the stomach itself; such ulcers do not often occur below the bile-duct, which supplies alkali to the intestine. The most interesting fact respecting ulceration here, is the observation of Mr Curling, that it often is met with in cases of burn; of this association he has cited numerous examples. We have now met with several such ulcers. A few of these burn-ulcers have been found elsewhere in the alimentary canal. We have seen one example in the stomach; and in one other of our cases, where a duodenal ulcer after a burn was fatal by hæmorrhage from the widely open pancreatico-duodenal artery, there was a deep eroded ulcer on the back of the tongue. Some of these duodenal ulcers from burn, go on to perforation and so kill by peritonitis.

Gall-stones sometimes ulcerate their way into the duodenum from an adherent gall-bladder.

You meet with great difference as regards the size of Brunner's glands in the duodenum; sometimes they are scarcely visible, at other times very large and prominent, as you see in this drawing, which was taken from a case of cholera. Sometimes one or more grow so large that they are described as "accessory pancreas."

Occasionally ulceration may be found existing through the whole of the small intestine, in the form of numerous ulcers with raised vascular edges, and without any disease in any other part of the body. These ulcers do not run in the course of the intestine, but their greatest length is from side to side, so as to entirely encompass the circumference of the bowel, if they do not quite meet, the incompleteness is on the side of attachment of the mesentery. Such cases are very rare, and when met with their pathology is not very clear.

The *jejunum* is almost free from primary disease, but we have twice met with clean cut perforating ulcers of the jejunum similar to "punched ulcer" of the stomach, there being no other disease present in the body, and the course of the cases being that of sudden and acute disease. Ulcers may be due to ileus from obstruction lower down.

We have met several times with severe local acute ileitis in the shape of a thickening of the whole of the coats, including the valvulæ conniventes which stood out stiffly, while the whole wall was thick with inflammatory lymph; the microscope showing a general charging of the whole tissue with pyoid corpuscles. This condition was found in a circumscribed patch of from six inches to two or three feet. Once the disease was uncomplicated; once a fatal condition in diabetes; once it arose in Bright's disease; and again, once in a yet severer form, going on to sloughing of the affected part, in the lardaceous intestine of an old syphilitic subject; a similar case is recorded by Dr Delafield also in lardaceous Bright's disease. In long-protracted diarrhœa some ulceration may be always looked for, except in children, where the diarrhœa or gastro-enteritis is often more an irritative effect of unsuitable food than a proper inflammation. In fatal cases of this kind we generally fail to discover any other appearance than an excessive enlargement of the intestinal glands; thus you may see the duodenal and Peyerian glands enlarged, and even more the solitary glands, both in the small and large intestine; these are seen as small bodies projecting from the mucous membrane. You should remember, however, that these glands, as well as the mesenteric, are comparatively larger in children than in adults; and thus we have heard students inquire if such have not been enlarged when we judged them to be only natural. You sometimes meet with *simple* follicular abscesses of the submucous coat, of a size from that of a lentil to that of a pea. These are usually found in scrofulous subjects, and then are seen as little sacs of pus; they are very different from the follicular *ulcers* which are associated with miliary tubercle in phthisis. We have not ourselves found tubercles secondarily developed around these follicular abscesses, though such an occurrence has been described.

Typhlitis, or inflammation of cæcum and appendix.—This is frequent and important, so as to require your special attention. When an inflammatory process has been confined to the coats of the gut itself, the term *cæcitis* or *typhlitis* has been given; and when the cellular tissue around is especially involved, *perityphlitis*. It is not clear, however, that any one particular form of disease is here intended by those who make use of those expressions. The cases to which these names are given frequently occur clinically, and recover; but when disease in the same region with similar characters proves fatal, we find usually some prior morbid process in the appendix, rather than in the cæcum itself. The appendix, again, may be the subject of ulceration in fever, phthisis, &c., as we have just seen; it may have various morbid secretions occurring in it, or concretions, or even foreign bodies lodging in it, which shall light up inflammation in the surrounding parts. The suddenness of the attacks of cæcitis, and the local peritonitis following, even in the large number of cases which recover, all point to the appendix as being the most frequent cause. Inflammations of the cæcum itself do occur, and sometimes are apparently caused by the continued lodgment of hard fæces in this part of the intestine. Such inflammation may, by ulcerating the mucous membrane, lead to perforation and local peritonitis, forming a fæcal abscess in the neighbourhood, which may discharge inwards, but we believe that this is comparatively rare.

In some cases it does not appear that an actual perforation has occurred; the inflammation may implicate the adjacent parts in the inflammatory process by contiguity. In those which recover, the inflammatory symptoms slowly subside, or an external suppuration may follow, and the cause remain unknown; and in fatal cases, so general a peritonitis may have occurred, or so much suppuration around the cæcum, that it is then difficult to arrive at the exact nature of the complaint; thus you may find the appendix sloughing or gangrenous, as if some foreign matter had been discharged from it, and yet be unable to discover it. Foreign bodies of small size, such as fruit-stones, may collect in the appendix, and cause its ulceration; and to show you how readily substances may enter it, here is a specimen, which many of you saw taken from a woman who lately died of hernia, and who, to obtain a passage through the bowels, swallowed a quantity of shot; these you see have lodged in the appendix. We believe, then, that many of the cases of disease supposed to affect the cæcum, arise rather from a chronic morbid process in the appendix. If you are in the habit of examining it, you may often find a morbid secretion within it (the mucous membrane possessing glands), a thick mucus, and, sometimes, a material like wax, which appears quite peculiar to it. The appendix is found rigid, and on opening it, is seen to contain a semi-translucent matter like wax, which on section often consists of concentric layers. This is sometimes of a brown colour, and hard, and at other times earthy matter is combined with it, until a perfect stony concretion or calculus is formed: it has a disposition to ulcerate its way through. Frequently, in cases that die after a sudden attack of peritonitis, this is found due to such a calculus, which had made its way through the appendix. Such cases are more common in young persons, especially of the male sex. In some cases the exterior of the concretion is earthy, and the centre soft and partly feculent. You will read in books that cherry-stones, and especially date-stones, have often accidentally lodged in the appendix and given rise to the subsequent mischief. We have never yet met with these bodies, but the hard brown concretions in the vermiform process very much resemble date-stones, so that one cannot help a suspicion that mistakes have often been made as to their character.

We may mention here some very curious cases where, in consequence of an apparently congenital smallness of the ileo-cæcal orifice, cherry-stones, &c., have accumulated in the end of the small intestine, producing most remarkable sensations and noises, almost justifying Mr Sawyer's account of the necklace. Such a case came under Sir W. Jenner's care, in the person of a middle-aged woman, whose collection of cherry-stones rattled and crackled very remarkably during clinical examination. After her death a pint of fruit-stones were found in the ileum, and the ileo-cæcal orifice was very small. According to her own account, she had never to her knowledge, when young, swallowed more cherry-stones than other children. The case, though very curious, is not unique; several such collections are on record.

Inflammation of large intestine.—The term colitis is sometimes used as though synonymous with dysentery. Our usual language has indeed been too indefinite, nay, incorrect, in speaking of all affections of the large intestine as dysenteric; for the true dysenteric process, although in many features like simple ulcerative colitis, yet is a disease having certain definite characters.

There is quite as much reason to regard febrile epidemic dysentery as a disease distinct from simple ulcerative colitis, as there is to regard febrile epidemic diphtheria as a disease distinct from croup. We have already seen how imperfect are the merely anatomical means of distinction between diphtheria and croup; and there is equal difficulty in distinguishing dysentery from simple colitis by anatomical characters. Dysentery, in fact, produces a variety of colitis distinguished from others by a special course, and by clinical phenomena; though when the dysenteric fever has passed off, the state of the colon which is left is much the same as that of simple ulceration.

Cases of true idiopathic colitis are rare, and stand, as we have said, in the same relation to true dysentery as croup does to diphtheria, from the absence of those peculiar features which give dysentery the characters of a specific fever. For example, we have seen a case attended by discharge of mucus and blood where, after death, the whole internal surface of the colon presented a highly vascular, soft, red surface, covered with tenacious mucus or adherent lymph, and here and there showing a few minute points of ulceration; the coats, also, were much swollen by exudation into the mucous and submucous tissues. In other examples there has been extensive ulceration, commencing in the follicles, and spreading from them to destroy the tissue around, thus producing a ragged ulcerated surface, which is so far identical with the effects of the specific fever dysentery, that the distinction between the two conditions rests on the characters of the accompanying constitutional disturbance, just as scarlatina is known from simple cynanche by the addition of its proper fever to the swelling of the tonsils.

Ulceration of the large intestine is one of the most common post-mortem appearances we meet with; in persons long ill with various visceral complaints you often will find large ulcers in various parts of the large bowel, but more especially in the cæcum, ascending colon, and sigmoid flexure. These are mostly of a chronic character, with raised indurated edges; some spreading while others are healing. They probably have the same pathology as many ulcers on the leg connected with a retardation of venous circulation. These ulcers have their length in the direction of the transverse course of the bowel, passing around its circumference. In cases of phthisis and tubercular disease, it is not uncommon to find such ulcers, and not, apparently, having a tuberculous character.

The rectum is very liable to be perforated by pelvic abscesses; also the cæcum by lumbar abscesses occasionally.

Pellicular or diphtheritic colitis.—That is the kind of inflammation where the secretion is firm, and forms an adherent layer on the surface. It is not unfrequently met with in the colon. On opening the large intestine, its whole inner surface appears of a grey colour, the mucous membrane being hidden by a firmly adherent deposit upon it; this can be stripped off with difficulty. It is found to be very tough, and its surface is granular; the mucous membrane beneath for the most part looks healthy, but in some places very minute ulcerations are present, occupying apparently the sites of the follicles, and in other parts it is highly congested. In these two specimens the lower end *of the ileum* was also involved, but only in patches; a stream of water *flowing* upon these failed to move the adherent lymph. A section shows the walls somewhat thickened, but this may be due to the contraction of the gut. We have seen this diphtheric colitis occur in two or three instances as an idiopathic and fatal form of disease. We have several times met with it along with a similar inflammation of the stomach ; also in Bright's disease. We have met with it, too, in diphtheria of the fauces, and a similar experience has happened to others. In these acute cases the adventitious matter could not be detached as a distinct membrane ; but occasionally we meet with chronic forms of pellicular inflammation of the large intestine, such that *casts* of the intestine may be passed as well as long pieces of mucus, which are often mistaken by the patient for worms. These long pieces consist mostly of mucus containing cells. Occasionally, if placed in water, they will be found to be hollow, and to correspond to the part of the gut from which they have exuded, constituting indeed a mould of the intestine, the microscope detecting on them the marks of the follicles, and of the arrangement of the mucous membrane. The patients who suffer from this condition generally exhibit great mental depression, and are amongst the most intractable of valetudinarian melancholics.

Fistula in Ano.—We may here mention the anatomical characters of *fistulæ in ano.* There is still a doubt whether these begin first in ulcers which perforate outwards, or in abscesses that burst inwards ; probably some arise in each of these ways. The history of pain at the commencement and sudden relief will signify the latter origin. We have traced some clearly to the suppuration of inflamed hæmorrhoidal veins. The fistula is found as a callous passage in the fatty tissue about, or in, the wall of the anal part of the rectum. It fails to heal because of the continuous suction-like action of the sphincter upon the ischio-rectal fossa, which tends to draw open any cavity there, just as the respiratory suction within the chest prevents the healing of phthisis. By far the larger number of fistulæ do not open higher than an inch and a half within the bowel, and these generally have their external orifices farther from the anal aperture. But those that run high often open near its margin. The internal communication may be at the top of the fistulous channel, but sometimes there is yet a sac above this opening, and sometimes the fistula forms branches and has several openings into the bowel ; the tissues about the anus are then dense and vascular. Fistulæ are more frequent in phthisical patients. We have twice seen tubercles in their walls under these circumstances. These phthisical fistulæ are generally wide and open at their ends so as to produce little pain, but sometimes the lower end is not so free as the upper, and then fæces entering at the time of defæcation create great suffering, which, however, is easily relieved by making the lower opening more free. Severe accidents rarely arise from operations on fistulæ, but we have seen once pyæmic abscess of the brain follow such an operation.

Enteric Fever.—Among the specific inflammations of the intestines there is none more interesting and important than the enteritis of typhoid or enteric fever. This shows itself in a peculiar affection of the solitary and agminated follicles, especially of the lower end of the ileum, so that they enlarge greatly as the tonsils do in scarlatina; indeed, the enlargement is identical with that of the scarlatinous tonsil in microscopic characters (Pl. VI), but there is usually more of destructive change in the glands in fever, probably due to the irritating contact of the intestinal contents; for it is greatest where the ileo-cæcal valve delays these contents.

This state of the glands is the pathognomonic feature of enteric fever; and although there are other dangers in the fever, yet the consequences of these changes in the bowel, say perforation or hæmorrhage, bring about a large proportion of the deaths from this disease. The swelling of Peyer's patches and the solitary glands is more severe in proportion to their nearness to the cæcum, so that you often find several Peyer's patches in the upper ileum or jejunum unaffected while those near the cæcum are destroyed. Still, if you examine carefully you will see that sometimes a higher patch is worse than a lower.

The appearance of the glands depends on the stage of the disease at which the examination is made; but in average cases, as of death early in the fourth week, you find close to the cæcum some patches destroyed and almost entirely removed, and higher up some swollen and partially destroyed; and higher still you find the disease limited to particular follicles in a patch, so that the latter has only a small spot or two of the affection. The history of the changes in the solitary and agminated glands is shortly this. These glands swell greatly, projecting like fungoid growths from the wall of the bowel. On section their structure is soft and medullary looking, and microscopic examination shows that the swelling is due to hypertrophic enlargement of the follicles, which have the structure of lymphatic glands (Pl. VI). The same change, however, invades the submucous tissue around and beneath, and we have seen the lymphomatous matter permeating the muscular and subperitoneal layers. Still its chief and primary seat is in the follicles and the tissue between them; and hence the disease culminates towards the death of this follicular layer and its separation *en masse* as a slough from its bed. The detachment is in a plane of demarcation which cuts the Peyer's patch off, generally from beneath first, and afterwards at its edges. Thus, at one stage it may be found attached only at its edges or only at a part of its edge, or during life it may be found in the fæcal discharges as a sequestrum cast off. Here is such a patch which was so found by Mr Herbert Smith, and which you see corresponds to this vacant Peyer's patch discovered afterwards by Mr Carey on the death of the patient. Often, however, the separation is piecemeal.

When dead and not detached such sloughs take up the yellow bile-pigment from the bowel contents, and look yellowish or brownish, producing a very characteristic appearance of the diseased patches, attached as they are around the "ulcers" laid bare by the partial separations already completed. In milder degrees of the affection the typhoid change may pass off without sloughing, the typhoid swelling being absorbed, as it always is in the mesenteric glands. These milder degrees are, as we have said, observed in the patches higher up the bowel. You would ask, Are these higher patches which thus appear less affected, really only later attacked, so that in time they would be as bad as the lower, or are they less severely rather than later attacked? The answer is that they do not go on to the graver disease observed below; for however late the death may be in the fever, you always find the gravest disease at the lower end of the ileum. For instance, perforation in the fifth or sixth week occurs in a lowest patch, and the upper patches are then less affected than ever. So late as this, indeed, you find all the patches completely free from typhoid sloughs, except in the rarer cases wherein the typhoid has relapsed, when you may find disease of evidently two dates. We have once met ulcers of three dates with a well-marked history of triple typhoid fever. In simple and usual cases of late perforation the swelling of the upper patches has almost or quite subsided, so that we can conclude the lesser affection of the higher patches at the twenty-first day to be through milder severity, and not earlier stage of the disease in them. The uppermost patches always entirely escape. At least, in the large number which we have inspected there was no case in which the uppermost patches were implicated. No one knows why there is this selection of the lower patches. May it be because the fæcal matters are detained at the ileo-cæcal valve? If this be true, it argues in favour of occasional laxatives in the course of the fever.

The peculiarities of the "typhoid ulcer," resulting from the separation of these sloughs, are quite sufficient to distinguish it from all other ulcers, even from tuberculous ulcerations, which also affect Peyer's patches. Thus: 1. The peculiar yellow slough will be found around and on the patches more or less loosely attached. 2. The ulcer left by detachment of the slough has a very thin floor, in which you generally see transverse muscular fibres, whereas the floor of a tuberculous ulcer is *always much thicker than the surrounding intestine*, however deep the ulcer may look; it also has tubercles in the subperitoneal tissue outside. 3. The affected parts hold to the figure and position of Peyer's patches; but tuberculous ulcers, when old, run transversely around the inside of the bowel. You must, however, note that in rare old cases of typhoid ulcer, as indeed in all long-standing ulceration of the bowel, the ulcer spreads transversely.

The very thin floor of a typhoid ulcer may slough or be perforated at one or several small holes, so that general peritonitis is set up by extravasation of fæces, or the same result may rarely be reached by extension of the inflammation through without sloughing or perforation. On the other hand, more than one patch may be perforated. In very rare cases the extravasated contents are encysted to form an abscess, which may then behave like other fæcal abscesses. Note that the situation in the abdomen where the perforated bowel lies is usually the right side of the pelvis, about its brim or just within it. The typhoid ulcer may open vessels, producing grave and perhaps fatal hæmorrhage.

Next you must notice that the process may be more severe in the large intestine, its solitary glands being affected like the solitary glands of the ileum, so that fatal perforation may occur there, especially in the cæcum. Even the cæcal appendix may suffer typhoid ulceration, as we have sometimes seen; and Dr Murchison has recorded a case in which it was perforated; so, too, we have seen a typhoid ulcer occur within a cæcal diverticulum of the ileum.

But, as we have hinted, death may occur from other conditions of the fever not belonging to the bowel; thus the patient may be overwhelmed by the fever in the blood as early as the eighth or tenth day, or earlier. Then you find in perfection the earlier stage of intumescence in the intestinal disease. It is about the tenth or twelfth day that you would see to perfection the glands thus affected; Peyer's patches presenting large raised oval masses projecting into the intestine, and amongst these, round masses like peas, corresponding to the solitary glands, which also are generally seen in the commencement of the colon; at this time the mesenteric glands are also found enlarged, congested, and softened, often forming tumour-like masses of some size. In other cases the cause of death may be bronchitis, &c., after the bowel disease has nearly healed, and then you find only healing ulcers in the intestine.

But besides peculiarities due to the *stage* of the disease, you will notice that sometimes the *degree* of affection in the bowel is remarkably slight, say with only half a dozen small spots affected characteristically. There are all degrees of severity, from this to a state wherein the interior of the lower twelve or eighteen inches of the ileum is converted into a hideous purple and grey sloughing pulp, of blood, and dying and dead tissue, while the mesenteric glands are nearly as bad. Of course, all the cases, light and severe looking, are equally dead, so that the fatality of the fever does not vary as the enteritis. A further proof of this you find in the converse cases, where patients following their ordinary avocations suffer typhoid perforation of the bowel while carrying a load in the street, &c. They were feeling poorly, but still able to work, while the bowel was so near perforation.

A distinct variety of typhoid patch is shown in the growth of it into bold fungous masses, of unusually firm texture, and with little tendency to slough. These are rare; they have been associated with syphilis. We ourselves have met with a very marked example of this form of typhoid affection in a man who had extreme syphilitic and lardaceous disease of the viscera.

The typhoid ulcers usually heal up very quickly. A month or two after the subsidence of fever there may be scarcely any discoverable trace of thickening or other deformity of the patches, &c.; and four months after, no sign of the typhoid except a partial deficiency of the patches.

Tubercular Disease and Ulceration of Intestine.—This is, perhaps, the commonest form of disease to which the intestine is liable, and it is remarkable that the same parts are affected as in fever, of which we have just been speaking. The disease is most frequently met with in conjunction with phthisis, and it is then that it can best be studied, since all its phases are constantly present. In an early stage all we can find is, on examining the lower end of the ileum, small, round, tuberculous deposits in the mucous membrane, corresponding mostly with the solitary glands; in the Peyerian patches also similar small round deposits may be found; at a later period these deposits are seen softening in their centre, and soon a small round excavation is formed, the *primary lenticular* ulcer; in the floor of this excavation other tubercles form, and opening into it compose a *secondary* or compound ulcer. The peculiarity of this process is, that as the ulcer deepens its floor thickens by new formation, so that perforation very rarely occurs. Only three times have we seen tubercular ulcers perforate the bowel; one of these was in the cæcal appendix, which is not unfrequently affected. Tubercles, however, still form in this thick base of the ulcer, extending into the subperitonal coat, where they very rarely indeed, however, set up tubercular peritonitis. They usually appear in the course of the lacteals to the mesenteric glands, in which usually secondary tubercles are found. These ulcers, like all long-standing ulcerations in the bowel, run transversely around the canal, forming at last broad raw bands. Sometimes healing occurs, but reopening of the ulcer is then likely. Occasionally, the puckering from the healing process may induce internal strangulation. This may result through the contraction of the ulcer in healing, and it may often take place when it is not quite healed. One such ulcer had contracted adhesion to the back of the bladder, and a loop of intestine became strangulated under it; the case being mistaken during the earlier stages for cystitis. Sometimes the coats of the intestine around and between the ulcers undergo acute inflammation, like the lung around vomicæ in pneumonic phthisis. We have already mentioned the small follicular abscesses in the coats of the intestine; these, though not tuberculous, generally occur in phthisis. Some regard them, in the present fashionable view, as casual non-tuberculous abscesses, which set up tubercle by infection of the parts around. This view, which amounts to regarding tubercle as a form of secondary chronic suppuration, is still under investigation. The commencement of the large intestine also generally contains similar ulcers, and a very favourite site for ulceration is the ileo-cæcal valve; also, if the appendix vermiformis be opened, some ulceration may be found within it. Sometimes you find tubercular ulceration of the colon, when the small intestine escapes.

Dysentery.—The disease bearing this name is believed by some, as the late Dr Baly, to commence by an affection of the solitary glands; this is the opinion of many of those who have seen the disease in tropical climates, although not the opinion of all. Some hold the simplest form of dysentery to be an inflammation of the whole mucous membrane, affecting especially the follicles, and leading to great swelling, vascularity, and secretion from the membrane, and subsequently ulceration, identical, indeed, with what we have already spoken of as simple colitis; but at other times dysentery appears in a croupous or diphtheritic form; and, therefore, in dysentery we may meet with a *simple inflammation* of the mucous membrane ending in ulceration, or a *diphtheritic inflammation*, or a disease beginning in the *solitary glands*. In the most rapid and fatal form there is no particularising such distinctions, but the mucous membrane becomes infiltrated with blood, and its tissue perishes either in great patches or in isolated spots. Thus, true epidemic dysentery includes all the inflammatory conditions of the large intestine, so that the diseases already described under the name of colitis are anatomically like dysentery, and we may look upon them as capable of passing into it under suitable epidemic conditions. In an acute form of the disease, then, you would see the mucous membrane of a deep red colour, swollen and pulpy, or soft, so that it can be easily torn, when some exudation would be found beneath it; the surface is covered with purulent mucus, and the solitary glands appear as numbers of small ulcers regularly placed. The ulcers grow more distinct and their sides become elevated by thickening, they are found especially on the summits of the rugosities of the bowel. The infiltration then advances farther, the ulcers becoming deeper, and the inflammation, perhaps, extends through the coats of the intestine, which swell to a thickness of several lines, and at the same time are soft. At this stage the peritoneum may be reached and peritonitis be set up, &c., or sloughing may occur with the same result. Microscopic examination of the tissue shows all the coats, but very especially the submucous, more or less crowded with wandering lymphoid cells, while the proper elements are cloudy and granular. If the person survive, the disease afterwards becomes chronic, and then the more elevated parts of the rugosities show deep ulcerations, while the parts that were in the recesses between the rugæ remain less affected; and thus the disease presents the appearance usually seen when coming under notice as a chronic affection in sailors who have arrived from abroad. In fatal cases of this kind, we find the colon extensively disorganised; the interior presenting a most irregular surface, from erosion by the ulcers; these may be isolated, and scattered over the membrane, but more usually they are united, and form a continuous serpentine ulceration throughout the large bowel, reminding in their distribution of the rugæ by the destruction of which they arose. The floor of these ulcers is the muscular coat, or, if any repair is going on, an adventitious hardened lymph which has formed on it. Between the ulcers, the mucous membrane is raised, red, and soft, in the form of so many islands. These may become wonderfully swollen and thickened, while they are still covered with the follicular mucous layer at their summits; they then look like crowds of polypi, and the condition is called *colitis polyposa*. If the curative process be at all present, the edges are vascular and indurated, the wall of the intestine is, at the same time, thickened by adventitious fibrous structure in the submucous tissue, and sometimes by hypertrophy of the muscular coat; the latter, however, may only be apparently thickened, owing to the contraction of the gut. These ulcers may heal, and then we find the various stages of cicatrisation present, or sometimes the intestine altogether healed; in the healing process a lymph is exuded on the floor of the ulcer, which becomes a hard callus, and which, contracting, draws together its walls, and thus you may often find cicatriform patches where the tissue appears puckered and radiating to a centre; when quite closed, all that is left is an unequal surface of a slate colour, these healed ulcers generally presenting a darker hue than the healthy mucous membrane, due, probably, to the change which the hæmatine has undergone; most of the pigmented changes found in membranes after inflammatory processes have their source in the chemical changes in the colouring matter of the blood. When a whole surface has been ulcerated and healed, it presents a very irregular and puckered aspect, and the hue is more or less of a slate colour. On the cicatrix you cannot find the beautiful areolar surface natural to the colon, which presents itself even to the naked eye as a minute stippling, and is very easily seen by a lens. To examine the state of the membrane, you should place a piece of the gut in a plate under water, when you will observe better the integrity or not of the surface. We have seen some cases where the ulcer has penetrated the colon, and set up a fatal peritonitis. The occurrence of hepatic abscess with dysentery we shall mention presently. Old dysenteric ulcers may reawaken into activity.

Syphilitic Ulceration.—In women the rectum is sometimes found ulcerated at the lowest part for a space of several inches, the ulcer, perhaps, running up into the sigmoid flexure. Such ulceration appears to depend on extension of inflammation along the perinæum in the primary stage of syphilis. The interior of the bowel is frightfully destroyed; there may be recto-vesical fistulæ, as in this specimen. The ulceration may pass in and out behind the mucous membrane and even deeper, so as to convert the wall of the bowel to very much the appearance of the apex of the right ventricle through the number of fleshy columns that cross it. The effect is a stricture of the rectum for which Amussat's operation may be required. Sometimes the bowel-wall is perforated and retro-peritoneal abscess is set up. Such ulceration of the rectum does not occur in males. Primary syphilitic sores are occasionally met with here.

Morbid Growths.—*Cancer.*—Secondary cancers of epithelial, carcinomatous, or sarcomatous kinds appear in the small as well as the large intestine, as little rounded patches seated in the mucous membrane; they are not unfrequent, but are apt to escape notice. We have known this form of cancer of the colon to be secondary to cancer of the liver, reversing the usual order of occurrence, in which the liver is infected from the colon. One also finds the intestine invaded and opened by cancer in neighbouring organs, thus the ileum, or, much more often, the rectum by uterine cancer; the colon by cancer of the stomach, gall-bladder, kidney, &c.

As to primary growths, the remarks we made on the varying degrees of malignancy, and therewith of softness of cancerous growths in the stomach, apply equally to such growths in the intestine.

Lymphoma.—You must note that the small intestine is very little subject to primary cancer, and when this does occur it nearly always has the form of lymphoma; that is, it has a structure almost identical with that of lymphatic gland (Pl. VI). It always then affects the mesenteric glands, enlarging them enormously, the tumour extending also to the lumbar glands; columns of the growth in the course of the lacteals generally connect the cancer in the bowel with the enlarged glands. This kind of cancer has three chief peculiarities:—First, it rarely ulcerates; second, it rather widens than narrows the channel of the bowel; and third, it shows very little disposition to undergo degenerative changes. To the naked eye it appears as a medullary cancer; its growth is rapid. Lymphoma may also affect the colon, but less frequently and less largely.

Lymphoma of the submucous coat is also found in association with leukæmia; it then has the same microscopic character as when the blood is unaffected, but it is usually in smaller patches and the glands less implicated.

In the *colon* primary cancer is frequent; it affects chiefly the cæcum or sigmoid flexure. One of the most interesting forms is *colloid*, which has here the same characters as in the stomach (p. 391). The gelatinous matter appears in some cases to be formed as an active outgrowth of the follicles of the mucous membrane. Colloid forms a thick mass of cancer, generally extending all around the bowel for several inches of its length; it may spread over and distend the peritoneum. On the other hand, sometimes the bowel is attacked by colloid which was primary in the peritoneum. It has less tendency to ulcerate and contract the bowel than carcinoma, but more than lymphoma.

Cylindrical epithelial cancer is relatively frequent in the large intestine, and may be combined with colloid; its structure is often a beautifully perfect follicular tissue, like that of Lieberkuhn's follicles, and its surface may grow into papillary processes; it may set up secondary cancer in the liver, which shall have exactly the same structure, that of intestinal mucous membrane. This disease in the colon is generally circumscribed at a small spot, and it constricts the bowel there. It is most frequent in the sigmoid flexure and rectum.

The more ordinary forms of *carcinoma* also occur in the intestines. Lymphoma, which we have already mentioned, would usually be called "medullary cancer;" but you meet also with true carcinoma in its medullary form as well as in its scirrhous form, and it is to these as well as to the colloid and epithelial forms that cancerous stricture is usually due, while, as we just now said, the lymphomatous variety of cancer widens the bowel. Carcinoma of the bowel has the same characters as carcinoma of the stomach (p. 389). In the intestine the fatal stricture need not be an entire closure. Often you are able to easily pass the blade of the enterotome through a stricture which has caused fatal obstruction. No doubt enemata are sometimes sent upwards past a stricture, with the effect of increasing the tension above it. In one case there could be no question that five pints of fluid passed up through a stricture which yet continued obstinate and proved fatal. The mucous surface of this form of cancer is constantly found as an ill-conditioned sore with thick raised edges. The ulceration may lead to perforation, with the usual consequences of this.

You sometimes find cancer at more than one point in the alimentary canal, as the duodenum and rectum. We have several times found the stomach and the colon separately affected in the same case.

Sometimes you may meet with *sarcoma*, especially in the form of melanosarcoma, secondary to melanosis of the eye or skin.

Tumours of the muscular tissue, or *myomata*, are said to occur and project inwards or outwards, but we have not observed this.

You meet occasionally with *villous cancer* in the intestine ; it has the same characters as in the stomach (p. 391). Also patches of superficial villous growth of simple fibrous structure with vascular loops ; these have no malignant character, they are called *papillary fibroma*. Such patches may arise in dysenteric ulcers.

Occasionally *lipoma* occurs in the submucous tissue of the bowel. It is generally in the form of little masses, which may project in a polypoid shape.

Cysts with mucous contents are found in the wall of the intestine ; they are very rare, but they sometimes reach a large size.

We have already spoken of the occurrence of tubercular ulceration. This is the only form in which tubercle attacks the intestines, for they generally escape or are very little affected in general miliary tuberculosis.

You occasionally meet with growths of a *polypoid* form hanging from the mucous membrane. Their seat is generally the rectum, where they come under the notice of the surgeon ; here are some which Mr Bryant removed from children. They are small round bodies, consisting of a fibro-cellular structure, and highly vascular. Sometimes, as in this specimen, they are composed of glandular tissue, like that of the mucous membrane itself ; they then resemble cylindrical epithelial cancer, but in the latter there is more of an interstitial texture loaded with active nuclei. Occasionally we meet with small polypoid growths in other parts of the canal, as you see in these specimens. In this one there are a number attached, forming a bunch of pendunculated tumours, very similar to those sometimes met with in the bladder. The solitary glands in the colon may enlarge and become pendulous.

Hæmorrhoids form a kind of tumour at and within the anal aperture. Their history is very simple. After death they generally look mild and innocent in comparison with the annoyance they have created during life ; they go down with the disappearance of the vital turgescence. Hæmorrhoids consist essentially of varicose swelling in the submucous tissue of the rectum and anus. The swollen veins there, are in connection with the proper venous system of the part, which you know is in free communication with the portal along the gut, and with the iliac system in the perinæum. The venous flow here has peculiar difficulties through gravitation, and through the pressure of fæces and of the internal genitalia, through irritation, &c., nay, even through muscular efforts of all kinds, for one cannot use his arms or even cough without bearing down upon the levator and sphincter ani, so as to check the freedom of venous current through those muscles. Hæmorrhoidal tumours are either all around the anus or limited to a part of it. They are either inside or outside, or partly in and partly out, and important surgical classifications for their treatment are based on their differences of site. But their pathological nature is always the same. They consist of dilated veins surrounded and separated by a lax fibrous tissue, and covered by mucous membrane or skin. It was held by some that the blood-cysts are cut off from the venous system; but this is not believed now, as under favorable circumstances injection from the veins can be effected. Sometimes the venules in them are numerous and tolerably even in size, at other times some are much larger than the rest, and at yet other times there is one great preponderating blood-cyst. Like other imperfections of the circulation, they are most apt to appear in adults as they are growing old; but sometimes they occur in the young, and they are sometimes hereditary or even congenital. Their chief importance arises from the accidents they are subject to. Thus, they are liable to be inflamed. As to this, Virchow lays stress on irritative annoyance by ill-conditioned or long-retained fæces, urging that the ordinary view of mechanical pressure on the veins does not include the whole cause of hæmorrhoidal congestion. Certainly inflammatory irritation makes up part of many attacks of "piles," and when sharp such inflammation may produce catarrh of their surface, and therewith much discomfort, especially if any ascarides casually present should take advantage of the moisture to make excursions in it as it oozes outwards. Bleeding from piles is frequent, and may be excessive in quantity; it sometimes arises evidently from the bursting of a varix, or it comes from the mucous membrane over the tumour; some think this most frequent; or else it may, as Mr Curling showed, be arterial from internal piles. Sometimes the internal piles coming down through the anus are grasped by the sphincter, and inflamed and strangulated. They then may slough; this is a very painful occurrence. At other times the blood in a pile will coagulate, and the thrombus will suppurate, and when the abscess is insufficiently opened a fistula may result. A certain number of anal fistulæ do so arise. Piles are limited to the lower end of the rectum, but we sometimes meet with a varicose condition extending some distance up the bowel.

Morbid Contents.—*Concretions.*—These may have been secreted in the intestinal canal, and are then called *enterolithes;* or they may have been formed from matters taken into the bowel, and then are named *accidental concretions.*

We have already mentioned that calculi may arise in the vermiform appendix; the earthy matter consisting mostly of phosphate of lime, which is secreted by the mucous membrane, and forms concentric layers; the nucleus being fæcal matter or some accidental substance. In other parts of the intestine very large calculi may arise, as you see here, and sometimes cause considerable obstruction; in this case the concretion made its way through from the cæcum. In the lower animals very large stones may form, as these from the intestine of a horse; they often have a nucleus of oats, or consist wholly of vegetable matters. In this one, composed of triple phosphate, the nucleus is a piece of iron. Some of the most remarkable of these are the bezoars, found in the intestines of the wild goat inhabiting the Persian province of Khorassan; these are soft, and contain a peculiar principle, called *ellagic acid*. *Accidental concretions* are those which form directly from substances taken as food or medicine, and thus masses of chalk or magnesia have occasionally collected in the inside; and we may here show you these remarkable hair-balls, like masses of felt, taken from the stomachs of cows, and a somewhat similar mass of hair we have seen removed from the stomach of a lunatic.

You may also meet with substances which have not been formed in the canal, but have passed into it, as gall-stones—these sometimes being so large as to cause fatal obstruction; several substances which did so, you will see on our shelves, as coins, knives, stones, &c.

Fæcal matters.—These are especially worthy of notice when discharged during life, but also on the post-mortem table you should observe their *form*, *consistence*, and *colour*. It is not altogether correct to say that solid fæcal matter is found only in the large intestine, for it may be seen of some consistence at the end of the small intestine. The fact of *scybala* being present, as well as the colour or fluidity of the fæcal matter, should be noticed as sometimes of importance: if of a dark colour, this may be due to blood, or to medicine taken, as iron; but sometimes a chemical test or the microscope is required to decide its origin. We have sometimes met with large quantities of hardened fæcal matter; thus, a round lump, weighing 17 oz., was in the sigmoid colon in one case; there was slight peritonitis over the colon at its seat. If blood has been poured out from the stomach it is of a black colour, but if it came from the lower part of the bowels it is of its natural bright hue, or of a brownish colour. The blood may have come from above the stomach; we have met with a case of fracture of the base of the skull with laceration of both carotids; the blood extended throughout the whole alimentary canal; the man was never insensible, and died of the bleeding. The presence or absence of bile should also now be observed, as well as during life.

Entozoa, or intestinal worms.—The four commonest kinds are the *tæniæ*, or tapeworms; *ascaris lumbricoides*, or round worm; *oxyuris* *vermicularis*, or thread-worm; and the *trichocephalus dispar*. The most interesting part of the history of intestinal worms is their mode of development, discovered by Von Siebold, Küchenmeister, Leuckart, and others; including in this country especially Dr Cobbold. These observers have shown that the vesicular worms, known as hydatids, are but the immature forms of tapeworms. The sexually complete animal, or tapeworm, inhabits the intestine, while the incomplete animal, in the form of an hydatid, is developed in a solid or parenchymatous organ. Thus, instead of the ova of the tapeworm producing young animals of the same form, an intermediate stage of development occurs, in which the head of the creature grows into its proper shape, and has a short neck, but the end of this develops into a large cyst, into which cyst the head and neck can retract themselves. The head and neck are known as the "*scolex*." Thus arises the cystic variety of worm, so that it is an incomplete form of the tapeworm. Experiments on the lower animals show that if a cysticercus be taken into the stomach it will soon be developed into a tænia; and again, that the ova which are produced from this animal, if taken into the intestine of another, are not formed into tæniæ there, but are taken up by the blood-vessels and deposited in the solid organs, as the liver, heart, brain, &c., where the first stage of development into a cyst occurs. Should they then be eaten with their host by another animal, the perfect worm is produced within the intestines of the devourer. It has thus been shown experimentally that the *cysticercus fasciolaris*, which inhabits the liver of the rat (a specimen of which you see here), if eaten by a cat, develops within her into a tapeworm. And again, that the ova of this tapeworm, if eaten by another rat, become cystic worms in its liver. In the same way the ova of the tænia cœnurus, which inhabits the intestine of the dog, passing out with the fæcal matter on to grass, may be eaten by sheep; the embryo is taken through the circulation into the brain of this animal, and there becomes the *cœnurus cerebralis;* this, if eaten by a dog, becomes again the tænia cœnurus. Experiments on the human subject have also shown that cysticerci may become developed into tæniæ; and it has been proved that it is the cysticercus of the pig which produces tænia solium; while tænia medio-canellata is produced from the cysticercus of the calf and ox.

The echinococcus is a cystic worm, which develops in the intestines of the dog or wolf into a small tapeworm with very few joints, but this tapeworm has, we believe, never been found in man. Although the difference in form between a round cyst and a long worm may appear great, yet such a specimen as the cysticercus of the rat will show how one may pass into the other; for you see it has a long neck, and so has already somewhat the appearance of a tapeworm; and then, as regards the form of the head, you know that in both there is a circlet of hooklets which enables the creature to fix itself to the neighbouring structure for the purpose of nutriment. The cysticercus cellulosæ, which is found in various parts of the human body, consists of an oval bag or cyst of thin membrane, containing within it a clear fluid, and from this cyst there protrudes a neck, which is of different lengths in different instances, and at the end of this is the head, which, like that of the tapeworm, has four suckers, and a circlet of hooks in the midst of them; the latter average twenty in number.

Tapeworms sometimes measure several feet in length. They are composed of a number of segments, each of these containing hermaphrodite sexual organs, and discharging innumerable ova. Although these are so far independent, they are all united, and gain their nourishment through the head, which is fixed to the intestinal wall of the host; from the other extreme the ripe segments drop off, like the fruit from a tree, whose root still remains in the form of the four suckers, fixed to the intestine. The head which is attached, is a little knob at the end of a long flat filament. This differs in different species; there are always in *tæniæ* four suckers, arranged equidistant around the raised centre of the free extremity. In *Tænia mediocanellata* the suckers are black and there are no hooklets, but in *T. solium* the suckers are pale, and there is in the centre between them a double circlet of hooklets concentrically placed, and circumscribing a small circle. Other varieties of Tænia, *T. marginata*, *T. elliptica*, have been known to occur, but very rarely. The tapeworm of the Swiss, *Bothriocephalus latus*, has a smooth oval end, with a groove on each side, but neither suckers nor hooklets. It is asserted by Knoch that this worm does not require an intermediate host in its development, but there are great doubts of the accuracy of this view. You will find numerous specimens of tapeworm on the shelves. Students often inquire, when patients bring portions of worms, whether these are from near the head or the opposite extremity. The head and neck consist of one long and very slender piece, and the segments which come next appear as very narrow pieces, then they increase in breadth until the middle of the worm is approached, when the breadth somewhat exceeds the length; towards the tail they become narrower and longer, each segment acquiring somewhat of a conical form, tapering to that extremity which is attached to the broad end of the segment before it. The place of growth of the ordinary tapeworm is the small intestine; you may find one after death occupying several feet of the intestine, stretched out at full length, the head directed upwards, but free and moving about as if seeking for some new place of attachment.

Trichocephalus dispar.—These are small hair-like worms, one or two inches long, the pointed end constituting the head, and they lie curled up as you see in these specimens. They are met with mostly after death in examining the intestines carefully, and it is said that in France nearly every body contains some in the cæcum. Although we have occasionally seen them, we cannot say that this is the case in England; indeed, they are somewhat rare here. This is a drawing of one which we examined not long ago; it is a male specimen, and shows at the blunt end the sexual organ projecting from a sheath. These worms are not known to produce any symptoms.

Ascaris lumbricoides is the common round worm resembling the earth-worm, and often called lumbricus; it is developed in the small intestine, where we not unfrequently meet with it in considerable numbers, especially in children; three hundred to five hundred are said to have been passed by one person. After death (as during life) the worm may pass into any part of the intestinal canal, as the stomach or œsophagus, or it may pass out from the mouth or nose. Cases are recorded where one got into the larynx and caused choking; also where the worm penetrated the intestine, causing fatal peritonitis, or pierced its way into the abdominal wall and there formed an abscess, or was found in the pleural cavity, or sometimes one of these worms has passed up the bile-duct.

Oxyuris vermicularis, the well-known thread worm, is developed in the rectum, sometimes in enormous numbers.

Ascaris mystax of the cat has been shown by Dr Cobbold to be occasionally found as a parasite in man.

For the anatomy of this and the other worms we must refer you to zoological works, especially the excellent monograph of Dr Cobbold. It appears that the ova of round worms pass out of the body, and are very slowly developed when they reach water; but it is yet uncertain how they obtain access to the human body, and especially whether an intermediate host is necessary.

Bilharzia hæmatobia, a parasite residing in the veins, though it more especially affects the urinary system, is found also in the intestines, where it produces extravasation of blood with thickening, ulceration, and fungoid projections arising in the mucous and submucous tissues. It is endemic in Egypt, and to a less extent at the Cape of Good Hope, producing a very terrible form of disease.

A minute fluke worm, less than a line long, distoma heterophyes, was found in hundreds by Dr Bilharz, of Cairo, in the intestines of a boy.

DISEASES OF THE LIVER

Malformation.—Sometimes the liver has an unusual shape, so that the whole organ is rounded or the relative size of the lobes is unnatural. It is often very doubtful whether such changes are not rather the result of bygone disease than of original malformation. We know from actual experience that the drying up of an abscess, or syphilitic deposit, &c., will very much alter the form of the liver in adult life; but we can infer that a much larger extent of change will arise from the like disease at earlier periods before growth is complete, and thus we should think that in a liver like this, which consists almost entirely of a large left lobe, the morbid condition that caused the atrophy of the other part must have been in action at a very early period of existence.

Sometimes an additional lobe is present in an otherwise quite healthy liver; we have seen it occasionally on the under surface of the right lobe. Projecting lobes, like these, are present very constantly in the livers of some kinds of animals, such as rodents, and yet the shape of the liver would appear to have no importance which would correspond to such constancy of its figure in any class of animals. Certainly the form of the liver is very commonly surprisingly altered by external influences, especially by tight lacing. When this is extreme, the right lobe may have its upper surface thrown into folds by the circumferential pressure, the convex surface yielding more easily than other parts. But the usual effect of tight lacing on the form of the liver is this—that a deep transverse furrow runs from side to side across the under surface of the right lobe; it is deepest at the right border, where it may divide the mass of the liver more or less definitely into two parts, one in front of the other. The liver, as you know, has naturally a somewhat bent figure, which is arched from before backwards, the arch fitting into the diaphragm. Circular pressure around the arch increases its convexity, bending it up in the middle, and compressing the middle part in the concavity of the arch, so that the substance here atrophies and wastes away, thus producing the transverse groove described. This groove may run across to the left lobe, and may be so deep as to completely divide the anterior from the posterior part, fibrous remains with vessels, &c., alone connecting the two portions. Thus, what appears to be a movable tumour in the region of the liver may be produced, for the organ is generally thrust down in these cases, so leading to serious errors in diagnosis. The groove itself is always more or less fibrous looking, in consequence of loss of the secreting tissue.

Men who wear straps round their waists suffer a degree of the same change, but in men usually the effects of this lesser pressure are different. A groove or depression with a fibrous appearance runs along above the anterior edge of the liver in front, where the false ribs press upon it, the whole of the thin front edge being also more fibrous than natural. The contrast between the anterior and posterior part in these cases in point of consistence is very remarkable in elderly persons. This sensitiveness of the liver to pressure clearly shows the necessity for its protection by the sheltered position which it naturally occupies. It is not uncommon to see on the left lobe a round patch of superficial fibrous wasting adjacent to the heart, and caused by the constant pounding of the liver by the heart against a flatulent or overfull stomach.

Hypertrophy.—The liver is often found to be large, and yet apparently healthy in texture. Analogy would lead us to expect such a result to arise from over-use of its function. We cannot say to what size the liver may reach from such a true hypertrophy; we have met with as much as 19 oz. of healthy kidney structure in persons who have drunk enormously of beer, and in similar examples have found the liver to reach 80 oz. weight, while its texture appeared healthy.

The term hypertrophy was formerly very loosely employed for any uniform enlargement of the liver; but we now recognise enlargements from engorgement, lardaceous disease, or other change, in the light of our knowledge of such diseases. The swelling which these produce is not an hypertrophy of liver tissue, but an introduction of some other matter into it. Simple hypertrophy is practically of little account; it is most certainly witnessed in cases where parts of the liver have wasted—for instance, in the tight-lace liver just described, where the groove is deepened by thickening of the rest of the organ; or when syphilitic or other local destruction has caused shrinking away of large parts, say not unfrequently of the entire left, or even of the right lobe, the remainder will become enormously enlarged. The lobules are then coarse; they appear to increase, and not to multiply. We have seen the whole right lobe removed, and the left then weighing 56 oz.

Atrophy.—The diseases that are characterised by extreme wasting of the liver tissue are so important that we must give careful attention to atrophies of the liver. Many of the diseases which we shall subsequently consider include more or less atrophy among their results; and in some, as in cirrhosis and the ramose atrophy of nutmeg liver (p. 432), this atrophy is an important part of all the change. Of this wasting we shall, however, speak in describing those kinds of disease.

The liver may be found simply wasted in cases such as cancer of the œsophagus, that is, where the cause of general wasting is of a simply starving kind. But there are other diseases, such as phthisis, in which, however wasted the whole body may be, the liver is commonly large; and this because the organ, such as the lung in phthisis, whose disorder leads to the wasting, gets help from the liver when its function is impaired, thus causing to the liver over-work and hypertrophy.

The diseases in which a simple atrophy of the liver forms the main characteristic feature are three, and are named, after the colour the liver shows, respectively—yellow, red, and brown.

Yellow atrophy and red atrophy.—The disease which includes these conditions is well defined, and yet anomalous, both clinically and anatomically. For though probably inflammatory and nearly always connected with unmistakable sub-inflammation, it nevertheless neither arises out of nor runs into the common forms of inflammation, viz. the ordinary chronic inflammation of cirrhosis and the intense inflammation of suppurative hepatitis; further, the capsule of the liver in acute yellow atrophy, as a general rule, is not inflamed, though acute inflammation of an organ commonly involves its capsule. Still, in occasional instances inflammation of the capsule is present, with general peritonitis; the bile-ducts and gall-bladder, also, while they vary a good deal in their state, often show a remarkable redness within, suggestive of inflammation, and they contain sometimes peculiar tenacious mucus.

Considering all these circumstances, we cannot set down yellow atrophy as unqualified acute inflammation, and yet, as we shall see, sub-inflammatory change around the portal vein causing red atrophy is so commonly, if not, as we believe, constantly met with in it, that the connection of yellow atrophy through red atrophy with inflammation must be held as undoubtedly made out.

The liver with yellow atrophy is small, often very small, weighing less than one half or even only one third of its normal weight. The organ is diminished equally in all directions, so as to preserve its natural form; but in consistence it is remarkably soft, supple, and flabby, the capsule wrinkling loosely from want of the natural bulk of its contents, and the organ when placed upon its edge doubling down upon itself, unable as it is to sustain its own weight. This doubling down may occur in the living body, the front of the liver falling back from the abdominal wall, while the colon rolls up into its place, taking entirely away the usual anterior area of hepatic dulness.

Some of these livers, however, have shown partial and unequal distributions of the yellow atrophy, intermediate parts being in the state of red atrophy, of which we shall presently speak. Under these circumstances the liver may be marked by uneven elevations, from the shrinking of the red atrophied parts out of proportion with the yellow. The outside is generally of a dirty greenish-clayey colour; but on section the colour is bright yellow, like moistened rhubarb, and in all the cases we have seen there has also been present some red atrophy in the section; this is found around the great vessels, especially but not only the portal veins. The outlines of the lobules on section, can scarcely be seen, either in the yellow or red part; but if seen at all are seen in the yellow part better than in the red, and the lobules are then seen to be much larger in the yellow than in the red. In those cases which have lasted longest the red portion is in greatest quantity; and this fact, taken with the microscopic characters of the two portions, shows that the red is a late stage, or chronic form, of the yellow atrophy, the yellow being an acute or excessive intensity of the red. A greyish material may also be seen between the lobules in the tracks of the portal venules; this is in an intermediate stage between the red and yellow stages, and signifies an incipient inflammatory thickening, of which the red condition is the end. There is nothing remarkable in the appearance of the channels of the blood-vessels, but it is said that injection will not run through the capillaries, breaking rather into the parenchyma of the lobules.

The microscopic appearance of the yellow atrophy is that which is best known; it reveals a destruction of the hepatic cells, which are found as granular relics in the lax and flocculent remains of the capillary network, little of the tissue structure remaining to be seen. But it is important to note that the yellow portion always shows some tolerably healthy lobules, and parts of lobules, whose cells persist, but are coloured bright yellow with bile-pigment. The process thus revealed is one of simple destruction of the secreting elements of the liver without any formative changes, this evidently being a rapid proceeding, corresponding, indeed, with the fearfully quick course of the grave symptoms in acute atrophy.

On the other hand, the red atrophy shows a very different appearance. The vascular network of the lobule is thickened much and shrunken, at the expense of the spaces which should contain the liver-cells. In these spaces small nucleated cells are found in parts, like epithelium; these appear to be degraded liver-cells, which have lost their proper character without perishing entirely; but for the most part the spaces contain only a few granular relics. We have seen in the thickened substance about the capillary network a quantity of pus-like cells, crowded in it around the portal capillaries. Thus, in the red part an inflammatory condition is proved to exist, not only by these pus-like cells in certain cases, but also by the thickening of the vascular stroma which resembles the thickening of it in cirrhosis; differing from this chiefly in two points, first, that it extends more uniformly throughout the lobules instead of being limited to the tracks of the portal vein, as in cirrhosis; and secondly, in the destruction of the liver-cells being greatly beyond what the pressure of this thickening will explain.

Besides these changes in the structural constitution of the liver, its chemical constitution is also changed. It may contain some fat, but generally this is present to no important extent. The peculiar character is in the presence of large quantities of leucin and tyrosin. The proper crystalline forms of these substances may be seen in the tissue, and may be found in the blood of the portal or hepatic vein. They may be absent in fresh specimens, and yet be found in large quantity when these specimens have been kept, with or without preservation in spirit. In warm weather we have seen a white efflorescence of these substances form on sections which have been for several hours exposed to the air.

The course of this disease is usually swift, lasting from three to five days, and characterised by a combination of grave cerebral symptoms with jaundice, hæmorrhages, and moderate fever. About two thirds of the patients are females, and of these many are pregnant. Intense emotional disturbances have appeared to cause it, and it has occurred during the fever of syphilis.

Besides the change in the liver, the heart and other muscles generally have their fibres in a state of fatty degeneration. The kidney also is commonly loaded with fat and deeply jaundiced, while crystals of tyrosin and leucin are present in it. Such a general distribution of fatty degeneration in the muscles, kidney, &c., is sometimes termed *steatosis*. The urine contains leucin and tyrosin.

In poisoning by phosphorus all the above-described changes of acute yellow atrophy have been found by German observers. In a case which came before us, however, the state of the liver was quite different, being so loaded with fat as almost to resemble tallow in appearance, and, indeed, in consistency also. Further observations of this interesting disease are still required. Some of the earliest-observed instances you will find in the first volume of the 'Guy's Hospital Reports' related by Dr Bright, and others in Dr Graves' lectures. But much light has been thrown since then upon its nature.

Brown atrophy.—By this we mean what has been called chronic atrophy by Frerichs, who described it as of a brown colour, and red atrophy by Rokitansky, who also says it is dark brown in colour. It is an entirely different kind of disease from the yellow and red atrophy we have just described, its symptoms being those of obstructed hepatic circulation without jaundice; symptoms, in fact identical with those of cirrhosis, but often accompanied with severe diarrhœa.

The liver is small; we have found it weighing only 28 oz., but it has been found only 24 oz. in weight. The organ is simply diminished in size, with perhaps its edge wasted and white looking. It is flabby, soft, but not so pulpy as in the yellow atrophy. There is no appearance of increase of fibrous tissue within or without, the capsule being thin, and the substance dark brownish-red—that is, of a deep liver colour. Microscopic examination shows the cells of the liver exceedingly small and charged with brown grains, many of which are set free by the bursting of the cells, the nuclei of which are generally plainly visible. Around the portal veins the microscope shows some thickening of Glisson's capsule, and we have seen on close observation of the section little, grey, tubercle-like points, microscopically composed of remains of liver tissue, in which all the veins were occluded. Attempts to inject the liver from the portal vein under these circumstances more or less entirely fail. There is a deficiency of bile. Brown atrophy has been known to occur in early life, but it generally appears about or after middle age, and it has been found associated with pigmented spleen from intermittent fever.

Congestion.—Being a highly vascular organ,—you know that microscopic sections of injected liver look as if there were nothing but blood-vessels in the liver,—the liver is subject to great congestion, which during life may enlarge it perceptibly, as you often observe in heart, or lung, or any ischæmic disease. At a post-mortem examination, however, a fulness of the liver with blood—that is, a simple congestion, proves nothing whatever. Mr Kiernan's several forms of liver congestion are only physiological amusements.

No doubt there is such congestion of the liver as is popularly understood, that is, a bloated state of the organ, produced either *mechanically*, by sedentary habits, or by more direct sources of obstruction; or *passively*, by torpid vein stream, suppressed discharges, &c.; or *actively*, by irritating foods, hot climates, miasms, fevers, and so forth. All this may be, we say; and so, no doubt, congestion is a useful medium of recognition for such cases during life. But after death such moderate congestion reveals simply nothing whatever, and we must remember, in thinking of these states, that they are very various and often complex, and the mere effect upon the blood-flow is a very minor consideration, although convenient in giving a common and well-understood name to the states which cause it.

The only congestions which we can recognise after death are the more intense *mechanical* congestions, and to these we wish to draw your attention. They arise from obstacles to the onflow of blood in the hepatic vein, producing an obstruction whose effect is to widen the main trunks of those veins and then their branches, until the capillaries of the hepatic veins, which you know are in the centres of the lobules, become distended. These capillaries, as you further know, converge from the circumference of the lobules, where they arise from the portal venules, gathering themselves together upon the intralobular hepatic venule which runs down the centre of the lobule.

Now, when this distension of the capillaries of the hepatic venules about the centres of the lobules has been only moderately severe and prolonged, they become dilated, and are not able to empty themselves even when the blood has got out of the great vessels in the removal of the liver from the body. Under these circumstances the section of the liver shows more or less dark centres to all the lobules, while the circumferences of the lobules are pale, for the capillaries near the portal vein are not thus distended, but anæmic, hence there arises a minute mottling, with dark and light parts more or less evident, and this state is common in death by chronic bronchitis or other diseases, wherein a varying and inconstant obstruction to blood-flow has existed.

Nutmeg liver.—But when this distension of the hepatic capillaries has been long in great force, as in fatal mitral obstruction, then the above-mentioned darkening of the centres of the lobules, contrasted with paleness of their circumference, becomes wonderfully strongly marked; the light and dark parts being sharply defined, so that the mixture of colours, without mingling, gives exactly the appearance of the cut surface of a nutmeg, the intricately folded and twisted lobulation revealed in the liver corresponding curiously with the similar twists and folds in a cut nutmeg.

A little more has, however, to be said before we fully understand the process.

The great and permanent distension of the hepatic capillaries, which you know are close set and radiating, presses severely on the columns of hepatic cells, which you know occupy the radiating spaces between the radiating vessels, and this pressure destroys gradually the hepatic cells, from the centres of the lobules outwards; while these capillaries widen much, and, containing dark blood, make a very black area on section. Meantime, whatever be the reason of it, the bile-pigment in the more central hepatic cells of the lobules is always in much greater quantity than at the circumference, perhaps because the distance which the bile has to travel to the bile-ducts on the surface of the lobule is greater from these centrally placed cells. Thus much for the dark central areas coloured by dark blood and bile-pigment. The white part, on the other hand, is always on the circumference of the lobule, and in it with any care you are sure to find the little line indicating the portal venule with its so-called Glisson's capsule around it. The portal capillaries, like arterial capillaries, to which they correspond, are empty of blood, whence the whiteness partly arises; but the whiteness is partly due to the presence of a quantity of fat in the liver-cells about the portal vein. It appears that the liver-cells have such an attraction for fat, that those nearest the supplying vessel take it all up and allow little to pass on to the deeper cells, over whom they have the advantage of position. Thus, the whiteness about the portal venules is due to anæmia and fat. A yellow tint is often present from engorgement of the bile-ducts, which you know goes so far as sometimes to cause jaundice in heart disease. In both the white and dark parts, but chiefly in the dark, the microscope often reveals blood-crystals lodged in the tissue, from bygone extravasations. And not unfrequently blood-black patches in the dependent part of the organ show more recent extravasations of blood, which may reach some size, and be called *apoplexy of the liver*.

Ramose atrophy.—If heart disease has been at once unusually long and severe, the effect on the liver may go yet further, and the tissue of the centres of the lobules may be reduced to a state like that of cavernous tissue, nothing but dilated blood-vessels remaining. We then find that the smaller hepatic veins surrounded by these dilated capillaries come to view on the section as deep red ramose lines, sunken on the section on account of the wasting around, and in many parts, the process of distension and destruction having extended quite through some lobules and segments of lobules, one sees shrunken patches, where in the midst of the red microscopically cavernous substance only little isolated portions of the white matter remain. The nutmeg appearance will then almost entirely have gone. Under these circumstances the organ is small, its surface uneven from the shrinking of parts, its capsule rather opaque, and often crowded with minute flocculent papilliform developments of the peritoneal textures. These little outgrowths are similar to the so-called Pacchionian bodies on the cerebral meninges, which, like them, are caused by chronic mechanical obstruction in the veins.

Hæmorrhage.—An excess of pressure on the blood-vessels from congestion may cause actual hæmorrhage into the tissue; you will find, however, that this is due, in most cases, to a disease of the blood itself, as in purpura, where there is a disposition to bleeding from all parts. As, however, one very frequent cause of this purpuric state is hepatic disease, hæmorrhage into the tissue will be most frequently found in the morbid organ, and if with this a diseased heart be present we have present a twofold cause for this condition. For in cases of morbus cordis you generally have a highly congested liver, the organ being in the state called "nutmeg liver," and sometimes (but more especially if the liver itself be diseased) you will find blood actually extravasated into the tissue, as we have just now said.

In order to prove that this is the case, you have simply to take the end of the water-pipe and insert it into the portal vein, when in a few minutes a stream of water will permeate the whole parenchyma of the liver, passing through the lobules into the hepatic veins, whence it may be seen coming out into the vena cava, at first red, but afterwards colourless. If now a section of the liver be made, it will be seen to be quite white, except in those parts where hæmorrhage into the tissue has occurred.

Fatty Liver.—We have already said that fat enters into the composition of "nutmeg liver." It is also very frequent in cirrhosis, &c. But the condition called fatty liver is that in which the whole organ is loaded with fat, this being its only or principal disease. A lesser degree of the same state can scarcely be called morbid, if we judge from the fact of fat being a normal element in the livers of many of the inferior animals; and also from the discovery of so much fat in the livers of persons who have died from some accidental causes, that we must suppose a great variety in the amount of fat may be constantly occurring. Thus we recently inspected the body of a butcher, who was killed by an ox, dying an hour after his injuries. In him we found a liver so fat that it floated on water, and yet he was thought by his friends to be in good health at the time of the accident. The proportion of fat in the healthy organ is only 2 or 3 per cent.; but what excess above this constitutes a morbid state we cannot say.

In a healthy liver you find this fat forming small granules in the hepatic cells; if it be in excess, they form globules, and in extreme cases the whole cell is filled with oil. In such instances the organ is much spread out and softened, and is of a pale colour, the edges being rounded as if the tissue were crammed to excess with the adventitious matter; at the same time it is of light specific gravity, so that, as in the case we had the other day, the liver may float when placed in water. Lesser degrees are constantly met with, which you will recognise after some experience by the appearance and touch; but there are various tests by which you discover the presence of fat: thus, the old and rough method, one, indeed, which we still employ, is to burn a piece of the liver; you must hold it in the lamp a sufficient length of time to allow the water to be driven out; while this is taking place, if fat be present, blue sparks are given off, and after a time the piece burns, and the oil drops from it, and may be collected on a piece of paper. You may also use chemical means: placing a piece of the organ in a tube with some hot ether, and then pouring the fluid out to solidify. But the best test is the microscope, for by this means you may tell to a great nicety the relative amount of fat present, and whether it predominates in one part more than another; you may find for example, small fat globules in one cell and large ones in another, until you discover some entirely filled with oil. Fat may appear to be free in the tissue; this is usually from breaking of the cells in preparation; but we have seen fat undoubtedly free in the liver texture.

In most cases of fatty liver which you examine you will find that the degeneration into, or increase of fat takes place on the circumference of the lobules, and thus, in viewing a section by transmitted light, a dark black margin is seen around each lobule; this is the rule and is almost invariable. We can hardly enumerate the conditions in which fatty liver may be met with; in phthisis you know it is frequently seen, so also it is often associated with scrofulous affections of other organs; and, we think, it may be said that in most long-standing diseases, especially when occurring in persons who have been long bedridden, a fatty liver may be expected; it may also be looked for in those who have too much indulged their appetites at table, and especially in men who have drunk largely of beer. Generally a fatty liver is considered to be secondary to other ailments, or at most associated with them in connection with a common cause, and, indeed, it still remains to be decided how far, when a fatty liver appears to be the most marked morbid condition of the body, it may be looked upon as the primary disorder. If so, it is extremely rare. There is, however, one cause of fatty liver which we must never forget; we refer to poisoning by phosphorus. We have already mentioned the fact that Continental observers have found yellow and red atrophy in this form of poisoning, which, by the way, is more frequent on the Continent. But in a case that fell under our own observation, in which jaundice appeared during a seven days' illness caused by taking phosphorous rat-poison, the liver was fat and much resembled lard in appearance and consistency (see Yellow Atrophy, page 427).

Lardaceous or Waxy Liver.—This peculiar morbid condition appears formerly to have been confounded with that of fatty change, and the more easily as it is sometimes associated with it and occurs in the same class of subjects—the cachectic and the tuberculous. At one time, you know, the recognised changes in organs were very few, and thus only cancer and tubercle were known as taking the place of the healthy tissues. It was afterwards found that fat might occupy them, then fibroid tissue, and now a lardaceous or wax-like substance, and so, indeed, there may be numerous other proximate principles formed in the body which are still to be discovered. The chemical nature of this substance is still doubtful, and we fear is likely to remain so—indeed, the difficulties that beset its chemical investigation do not seem to have been realised plainly enough by those who have published analyses of it.

Two things have to be remembered and somehow overcome before its composition is settled: first, it is not a free deposit which can be gathered out of the texture by mechanical means, but rather, as we shall see, the new matter is *in the muscle cells of the small arteries*, and in the walls of the capillaries, and perhaps within the ultimate gland cells; secondly, no solvent of it has yet been found; it is remarkably callous to chemical solvents.

So, in truth, no one has ever obtained lardaceous matter for chemical examination, and the chemical analyses published, which state that it cannot be starchy because it contains nitrogenized matter, add nothing to our information respecting it; for whatever the substance may be, the parts examined included the nitrogenous tissue wherein the substance lies.

Dr Dickinson says that dealkalised fibrin—that is, fibrin obtained from an acid solution—shows the same reactions as the lardaceous viscera, and he has constructed a theory of the formation of the disease on the foundation he thus lays down. Shortly, the theory is much as follows:

Firstly, those people who have lardaceous viscera mostly have been the subjects of long-standing purulent discharge; secondly, pus is alkaline, and so carries off much alkali; hence he thinks we may believe that the long-enduring discharge of pus would carry off alkali enough to dealkalise some of the fibrin in the body, and that this fibrin is then deposited as lardaceous matter. He proposes to call the disease *depurative*, on account of the loss of pus which he believes to occasion it. The theory is very ingenious, but unsatisfactory in many ways; for in some of these cases no discharge of pus has existed; and, further, no explanation is worth much to us which does not throw light on the curious distribution of the diseased material which is practically limited to certain digesting viscera, the kidneys, and the lymphatic glands; parts which, you will see, have no direct functional connection. Indeed, almost the only thing that is even now known about the lardaceous matter is the curious attraction for iodine which it shows, and the deep walnut stain which iodine produces upon it. This walnut-colour is seen under the microscope to change to blue on the addition of sulphuric acid. These reactions closely resemble those of certain starchy matters, and it appears to us useful to retain the term *amyloid*, at least, for the reaction which characterises the substance. Whatever this amyloid matter may be, no doubt it is in no way at all like scrofulous or tuberculous products, for these consist of new-formed cellular elements; whereas in lardaceous disease there are no new cells, but the morbid substance is deposited within the existing elements of the tissue.

Snuff

As yet the microscope has revealed nearly all that we accurately know about the amyloid substance; it shows us its site in the tissues, and also its peculiarities of appearance; for those elements of the tissue which are diseased are seen to be swollen with a highly refractile, clear, lustrous, resin-like appearance. But the outlines of the elements affected are obscured through the great refractility of the amyloid material.

A most interesting relation to the small arteries is seen; the inner coats of these are so affected that the arteries look like clear translucent tubes. The muscle-cells in the wall of the artery are certainly involved, and, no doubt, the lining membrane also; at least, this is probable, because the disease extends on into the capillaries, producing in them the same swollen lustrous appearance. On the other hand, the large arteries are never affected.

It appears that the amyloid matter extends from the vessels into the proper secreting cells of the liver, altering them in the same way to lustrous translucent little bodies, in which no nucleus can be seen. But the change does not affect all parts of the hepatic lobule together; it begins at those parts into which the hepatic artery more immediately enters. This is usually in the middle zone of the lobule, neither at its centre nor at its circumference. By comparing this with what you find in fatty and pigmented livers, you will see that as fat usually occupies the outer zone, and excess of pigment is found chiefly at the centre, these three kinds of morbid deposit have their several especial seats, thus revealing a difference in the habit of these tracts in the lobule, though all parts of it have the same appearance when healthy.

We must next observe that though the distribution of disease in the liver is generally uniform, or nearly so, yet sometimes it is partial and sometimes very unequal, so that portions of the organ show a yellowish-white colour, and may resemble at first sight syphilitic patches; but we have never seen any shrivelling result from this. Lardaceous disease enlarges the part affected, and we think it does not lead to shrinking nor to a cicatricial appearance, as some have said.

The liver may be more or less infiltrated with the adventitious matter; when slightly so, the microscope may be required to recognise it; but when the quantity is excessive, the liver becomes enormously enlarged, almost filling the abdomen, and acquires great weight; thus, a short time ago a liver of this kind weighed fourteen pounds. It is dense and very inelastic. Unless fat be also present in it, as it often is, the liver has its usual shape and sharpness of edge, but if there is much fat the edges are rounded. The organ, however, tends to mould itself upon the solid organs it touches, the posterior part being hollowed for the kidney; and if, as is often the case, it has reached far to the left side, there may be also a concavity for the spleen. Instead of feeling soft, it is hard and firm, and the peculiar sensation given to the knife when cutting it is very characteristic, like that of raw bacon, whence its name. Thus, it cuts firm, so that the thinnest slices may be taken off with ease, and if these be held to the light they will be found translucent, much like a tissue that has been placed in glycerine. You will be able clearly to make out the lobules of such a lardaceous liver; you know that these lobules do not, as in some of the lower animals, form really distinct portions of the tissue, but are appearances due merely to the mode of distribution and connection of the blood-vessels; they are, however, remarkably well defined in the lardaceous organ. The tissue is peculiarly dry, and on pressure, only a little watery pink blood escapes from the vessels. If the organ be only partially infiltrated, so that it is not yet evidently enlarged, the disease may be recognised by the increased weight and inelastic hardness given it; thus, so different is it from the fatty liver we met with the other day, which floated in water, that portions of lardaceous liver we have found to be of a specific gravity of 1084.

These lardaceous livers are found mostly in scrofulous persons, and especially in the young who have suffered a long time with disease of the bones, whence it has been suggested whether any gelatiniform material might have been taken up from the osseous system and carried through the body. But the bone itself in these cases does not give the amyloid reaction, so there is little foundation for that belief. Phthisis is another frequent cause, but only chronic phthisis, such as is called "fibroid." Indeed, any form of prolonged suppuration may bring it on. Twice we have seen it from old carbuncles; once from leprosy, and occasionally from cancerous sores. Syphilis without any suppuration will certainly cause it, so that it is not brought about exclusively by discharge of pus. The cases in which it has been set down to intermittent fevers appear all to have been complicated with syphilis.

Inflammation.—In speaking of acute yellow atrophy of the liver, we have mentioned that the shrinking of the liver in yellow and red atrophy is of an irritant kind, but that it does not usually produce inflammation of the capsule or other signs of thorough inflammation. In these conditions the inflammation is so held under restraint, as it were, by the specific nature of the disease, that it does not reach the conditions proper to a characteristic degree of inflammatory disorder. The inflammations of the liver, as usually recognised, are acute or chronic. The only certainly recognised acute inflammation of the tissue of the liver in this country is that which leads to suppuration, and which is commonly called abscess of the liver. We have no anatomical evidence of any state of liver corresponding to acute Bright's disease in the kidney, or acute pneumonia in the lung, and this fact suggests that we may be overlooking important diseases of the liver.

Irritative swelling.—You must not, however, fail to notice the state of the liver in grave febrile disease, such as typhus, pyæmia, &c. In these diseases all the organs are bloated and soft, and loaded with blood, and the liver partakes in these changes very largely. It is easy to let this condition of the liver pass for a secondary state due to febrile blood-change in the same way as we so regard the softened spleen; but some observers are disposed to give these livers more particular significance, and, in short, consider them to be truly inflamed. The state of liver to which we are alluding shows itself in a moderate swelling of the organ with a loss of its natural consistence, so that it is more pliable and soft and less elastic than is natural. The section-face generally has a dull shine, like cut moist clay; it is paler than natural; in fact, its look is often quite remarkable. The microscopic appearances are not so decidedly abnormal as the above characters might lead us to expect. Considerable changes have been described by some. Thus it is said that the liver-cells are swollen and granular, being, in short, in the condition termed by Virchow "cloudy swelling;" but this is a state not easy to recognise with certainty in a liver, as its cells are naturally cloudy and variable. It is further said that exudation-cells can be seen about the portal veins, and in the lobule-tissue near them. This, however, is certainly not a constant appearance. What is most constant is an evident enlargement of the lobules, explaining the swelling which the naked eye already sees; and further, there is evidently a soaked or œdematous swollen appearance of all the interstitial connective substances.

Before, however, we can include this condition among true inflammations we should require some evidence to prove a tendency on its part to run on into more certain inflammation. Instead of this, however, the capsule of such a liver is never inflamed, and no evidence has been brought to show that prolonged or severe cases of fever lead into inflammatory hepatic changes, such as abscess or cirrhosis of liver. Hence the word irritation appears better to express what we know of it. Meantime this condition is no doubt very important among the lesions, which bring dangers in fevers, and a better knowledge of it is much to be desired.

Acute hepatitis.—Inflammation may be chronic or acute; we know little of hepatitis in its acute form in this country, except in its results as abscess, and it is questionable whether it ever occurs as an idiopathic disease in a temperate climate. In cases of suppuration, the adjacent tissue may be found in various stages of inflammation, and an hepatitis, we believe, exists during the early pyrexia, and precedes the formation of pus; but this stage is short, and is rarely seen, perhaps never in this country as an *idiopathic* affection. In tropical climates, however, such an hepatitis is a common affection, leading to various results, as abscess or gangrene; the former may be cured. Or the inflammation, in the tropics, may be more chronic and lead to cirrhosis.

Abscess.—This is occasionally met with arising from injury; thus a blow producing fracture of the ribs on the right side we have seen cause an hepatic abscess, and more than once produce a suppuration between the liver and diaphragm, which involved the former.

But hepatic abscess usually arises from pyæmia, either when this is of a general character, but much more often from a local pyæmia affecting the portal veins in connection with dysentery. In *general pyæmia*, as in the instance where death follows an amputation, abscesses may form in any part of the body. As a rule, these are confined to the lungs, and only in exceptional cases is the liver affected. We have, however, very repeatedly seen large abscesses in the liver, which sometimes have produced fatal peritonitis by bursting. It was stated by Pott, and since repeated, that such abscesses are more frequent after injuries of the head, but this we cannot corroborate from memory and without searching through the records, although we think it is true. It is thought that some of the elements of pus or putrid matter are taken into a vein and carried to the lungs, where they produce a peculiar form of lobular inflammation, which we have already described; and that if any of these elements should be carried into the general circulation, the other organs, as the liver, may be involved. The difficulty in understanding this process is, that the liver sometimes may be the only organ affected, or the suppuration is evidently older than that in the lung. We have, however, only met with two or three instances of this; in one, it was probable that a blow had been received over the liver; and in another there was suppuration in the pelvis, which might have given an independent source for the purulent infection.

Besides these cases, arising from general pyæmia and injury, there is another class which is associated with dysentery, and which is supposed to be dependent on the intestinal disease, or a *local pyæmia*. Dr Budd observed that the majority of hepatic abscesses we meet with are in the persons of sailors who have been in the tropics; and in whom on post-mortem examination old ulcers are found in the colon, from which fact he was led to believe that a morbid material had been taken up from the ulcers by the portal vein, and had set up inflammation and abscess in the liver.

There can be no doubt that the observations of Dr Budd are correct, since our own and other records corroborate them. These show that, excepting those cases of hepatic abscess which stand in connection with the two causes just named, nearly all the examples of that condition are in persons from the tropics, and are associated with dysentery, for on looking through our cases, we find only one of abscess in the liver which can be designated by the name of idiopathic, and even here there was the history of a blow preceding the symptoms. But in these cases, although the liver and the colon are affected, it is a question, Which affection stands to the other in the position of cause, and which of effect? The difficulty sometimes arises through the apparently recent state of the colic ulceration as compared with the chronic nature of the abscess in the liver, but more especially through the fact of the two occurring together only under certain circumstances. Thus no affection is more common in this country than ulceration of the intestine, and yet it is rarely followed by hepatic abscess. Moreover, in some parts of Europe a true dysentery occurs, and yet no hepatic affection. Thus, a few years ago several hundred bodies were examined at Prague, after death by dysentery, and yet in not one was the liver affected. Also in Norway a severe epidemic occurred with many deaths, and the only sequelæ observed were ascites and acute rheumatism. It is chiefly in tropical climates where hepatitis occurs that the connection is found, and therefore it seems at first more rational to attribute the hepatitis to some other cause than to the intestinal affection. Thus, in India it has been observed by our own surgeons, as well as by the French in Algeria, that during certain epidemics a number of soldiers will have dysentery, some hepatic abscess, and others the two together; in the latter case the doctors seem more inclined to attribute the colitis to the hepatic abscess, than *vice versâ*. We believe, however, the fact still remains, that in persons coming from abroad with hepatic abscess, an ulcerated colon is almost sure to be discovered, or the cicatrices of ulcers; at least, our own experience in this respect corroborates Dr Budd's.

Also it must be said that the experience given by those who have met with numerous cases of hepatic abscess in the tropics differs somewhat from Dr Budd's statement. Thus in as many as one fourth of 204 cases no traces of ulceration could be found in the intestine, though the patient had died of hepatic abscess. And Mr Waring, who adduces these cases, states, as another important contradiction to Dr Budd's view, that of 300 cases of hepatic abscess which proved fatal in India little more than one fourth had been preceded by symptoms of dysentery.

But we must remember in estimating the value of evidence from such large series of cases that the non-discovery of an ulcer is not a positive but a negative result, and it is surely probable that some inspections out of so many would be made hastily or under unfavorable circumstances, and scars of ulcer in the bowel are easily overlooked, while the whole of the tract that might furnish place for an ulcer is extremely long, requiring much more than average care for a thorough search. Hence we think that the non-discovery of ulceration in one fourth only of so many cases is by no means very trustworthy and conclusive proof of their non-existence. The most important positive evidence adduced against Dr Budd's view appears to us to be that brought by Dr Murchison, who points out a difference in the size and number of the liver abscesses which occur abroad and at home. Tropical abscesses are generally single and large, many abscesses in a liver being decidedly rare, while pyæmic abscesses, as we meet them at home, for the most part are small, and many are present scattered about in the organ close to its surface. Dr Murchison's own experience will enable him to speak with a knowledge of both the sorts of abscess he describes. We must not forget, however, that common pyæmic abscess of the liver in England being part of a *general* pyæmia is comparatively quickly fatal, so that the abscesses have little time to enlarge and break into each other. These abscesses, further, are prone to occur in the hinder part of the right lobe, the part which lies lowest when the patient is on his back. And in cases of some standing we have seen them breaking into each other as though a large collection would have in time resulted. Tropical abscesses, on the other hand, generally run a longer course in which to enlarge, and to unite if originally multiple; they also by preference affect the hinder part of the right lobe. In fact, the liver-abscess of this country which occurs in pyæmia is generally overlooked during the life of the patient, whose troubles from coexistent abscesses in the lungs, joints, &c., together making up the sad compendium which we call pyæmia, reduce the hepatic suppuration to the dimensions of a weighty addition to an already desperate evil. We have never known abscess of the liver resulting from general pyæmia in this country to have so much time allowed it, or to be so far the solitary mischief as to enable it to become an object for special treatment. Herein arises a great and most important practical distinction from the tropical abscess, which may become chronic because it often is the only very grave disease present. Indeed, it may when very large be wonderfully secret; no pain and scarce any hectic disturbing the patient. These large chronic tropical abscesses are those which afford opportunities for successful treatment, and in some 20 per cent. allow of cure. Generally they are single, and rarely more than two are present. The differences between tropical and pyæmic abscesses may then, we think, depend on their different age.

Next let us pass on to consider the stages of formation to be observed in hepatic abscesses. These are, of course, best known in the pyæmic abscess, having been studied in this country and in Germany by professed pathologists, and with much care. The opinion regarding them which at present prevails among what may be called the most advanced pathologists is sufficiently expressed by the name "embolic," which has been given them. The history of the process on the embolic theory is engagingly simple; it can be understood on purely mechanical considerations. Shortly it is this: A portion of clot from some vein in the primary ulcer, the source of the mischief, is carried into a branch of the portal vein, where it lodges and cuts off the blood supply from a certain area corresponding to the part the starved portion should supply. The capillaries on the confines of the territories around dilate; but yet though they bring blood into the starved part the natural *vis a tergo* which should come down the obstructed vessel is stopped by the clot, and hence the blood in these dilated capillaries stagnates. Herein arises the first stage of congestion, characterised by great engorgement of the affected patch of liver, especially of its circumferential part.

We should, in passing, notice a difficulty which is sure to arise in the student's mind. He would naturally reason thus: The portal vein brings blood to the liver in all its parts; if a branch of the portal vein be blocked, so far the part of the liver it should supply will not have blood brought to it, and hence will be anæmic. But this consideration is outweighed, as our description has already inferred, by the fact that blood can readily enter in spite of the portal obstruction, either through the hepatic artery or the surrounding branches of the portal vein, or by reflux through the hepatic veins; from these sources blood enters the area which is cut off by the obstruction. But the blood so entering is deprived by the obstruction, of the natural force in the portal vein, which should drive it on, and thus it comes to form a sort of eddy or pool, around which the current is free while it itself is turgid and stagnant.

The next stage in the process is put by some advanced pathologists in the following simple form: They say the obstructed territory of the liver cut off from its natural circulation of blood, proceeds to perish; dying and undergoing separation from the part around, as dead tissues sooner or later invariably do, by the sequestering process. The disintegrated dead portion lies in the cavity formed by its separation, mixed with pus secreted by the surrounding wall of living tissue, which in sufficient time is converted into the usual abscess wall or "pyogenic membrane." You will perceive that this description corresponds exactly with what is stated to occur in the lung also through embolism; but we have already shown in the case of the lung that clots obstructing the artery do not lead to such death of the obstructed part, unless the plug comes from some severely inflamed part. In the case of the liver the proof of this is less plentifully offered than in the pathology of the lungs; yet there is plenty of evidence to afford ample proof that mechanical obstruction of the portal veins will not cause death of the obstructed portion. Thus we have several times seen branches of the portal veins stuffed with cancer brought in from cancer of the stomach or intestine, and we have also seen all or part of the portal vein entirely obstructed in cases of cirrhosis, but in these cases no trace of any approach to death of the obstructed part occurred. Hence we think that in the liver, as in the lung, embolism of the supplying vessels does not lead to abscess through simply mechanical consequences, but rather that the irritant quality residing in the clot from the inflamed region and belonging to it as part of the products of inflammation there, forms the determining cause of the suppuration that arises when such a clot enters the liver. Still we must not leave the mechanical effects out of consideration; it cannot but arise that the dilatation of the vessels with stagnation of their contents must compress and weaken the liver tissue, and prepare it to yield to the destructive influence of the inflammatory cause. The first stage, then, an embolic abscess shows to the naked eye is swelling and reddening of the affected part, whose structure looks hazy and watery, the redness being most marked around the margin.

The second stage shows a breaking down of the portion involved, with every appearance of rapid disintegration into a purulent fluid. We must, however, note that there is usually no sign of gangrene, no foul decomposition, that is, in the patch, such as the actual death and separation which the mechanical explanation we have referred to would lead us to expect. An actual gangrene, foul smelling and decomposing, though it does occur in the liver, is certainly rare; it is always connected with a gangrenous condition of the primary ulcer or wound which furnishes the source of the pyæmia.

In the next stage the abscess completes itself and may discharge its contents by bursting. This occurrence in pyæmic or embolic abscess is rare, death coming early and even usually before the abscess has become rounded or acquired a smooth interior, so that we find it enclosed by the more or less ragged remains of the doomed tissue, as yet not entirely destroyed. Nevertheless, in a few cases these English pyæmic abscesses do burst, usually into the peritoneal cavity. You should always examine the veins of the liver around the acute abscesses; you will then often find that the pus has entered a portal or hepatic vein to some length; it may even thus extend into the right heart; the pus is then separated from the blood by a film of yellow fibrin.

The large abscesses which Dr Murchison calls tropical, and which usually come from hot climates, are of older standing, so that they are generally more or less encysted; that is, the wall of the abscess is tough and fibrous, like a special cyst enclosing the pus; the cyst arising through a thickening of the natural fibrous tissue present in the implicated part of the liver. The pressure and the always destructive influence of the near neighbouring inflammation will have done away with the secreting tissue adjoining to the pus, while the fibrous constituents persist being naturally more hardy. Further, they not only persist, but become increased as fibrous tissues always do when long exposed to, or participating in, inflammation. The colour of the contents varies, being greenish-cream-colour when pure, but often getting various tints of yellow or brown through mixture with bile or altered blood. Such colours of the pus you should take care to remember when you see them, for we have known the character of the pus lead to correct diagnosis when an abscess of the liver has burst through the diaphragm into the lung and been spat up; patients have been known to complain of the bitter taste of such pus.

It is important to be aware of the various modes of termination in different examples of tropical abscess of the liver. They may exhaust the patient, and kill without reaching the surface; and they may diminish and shrivel, the cyst contracting around them, until only a pasty, or putty-like, or white dry matter is left. But either of these courses is exceptional, and indeed rare; several cases have made it nearly certain that even when they have lain quiet for a long while unsuspected, they may increase and become active again; the patient, who has been years home from India, seeming to be after the long interval attacked with hepatic abscess. But the more ordinary course of such abscesses is to discharge themselves within a few months of their first formation, the direction in which the matter escapes depending on the position it occupies. It may go into any of the cavities to which the liver is adjacent, such as the pleura, pericardium, colon, stomach, duodenum, gall bladder, or even the pelvis of the kidney. To reach any of these, except the last two, the peritoneal surfaces must become adherent prior to the perforation. Should such adhesion not occur, the abscess may burst into the peritoneum. Or passing through the diaphragm it may find the lung unadherent, so bursting into the pleural cavity; or the inflammation may cause adhesion of the lung, when the abscess bursts into the lung tissue and reaches the bronchial tubes. As the abscesses are usually in the right lobe and near the surface, it will easily be inferred that the most frequent way of escape is through the diaphragm upwards, or to the surface of the body outwards, seeing that by far the larger part of the right lobe is separated by the diaphragm from the lung and parietes. Hence these ways of escape are the most frequent; but you must remember that when the abscess comes through the abdominal wall, which is the most favourable course it can pursue, the fasciæ and the tendons cause it to take an irregular and often a long course, burrowing in the substance of the abdominal wall, and making its appearance at some distance from the real seat of the abscess. The surgeon, however, usually is called to interfere, and makes the straightest way for it he can.

While speaking of abscess in the tissue of the liver, we must bear in mind that a hydatid cyst may suppurate, so forming an abscess in which, of course, the bodies of the hydatids will be seen, unless the case has lasted so long that they are disintegrated. Some long-standing abscesses may, perhaps, be of this nature.

Besides the pyæmic abscesses due to embolism, and the tropical abscesses which fall doubtfully under the same kind, there are other suppurations in which the liver is concerned, though by far more rarely, especially *suppuration of the portal vein*, and abscess outside upon the liver, or "*Perihepatic abscess.*"

Suppuration of the portal vein.—Sometimes this takes a very definite form; the portal vein-trunk and its branches being bathed in pus, and their channel obstructed with blood-clot, which is more or less altered, and generally contains pus; or the channel may be open, for a greater or less extent, into the suppurating space around. This is not a frequent form of disease, but many cases are recorded; some ulceration or suppuration in the field of origin of the portal system will be nearly always found to have given rise to the portal suppuration, or *pylephlebitis*, as it is called. Thus, in a case we had recently under observation, we found abscesses in the submucous tissue of the rectum, which so implicated the hæmorrhoidal veins, and extended up the inferior mesenteric vein as to reach the portal vein, around whose trunk there was extensive suppuration, spreading irregularly into the liver along its branches. The disease which gives rise to such suppuration may be in the alimentary canal, gall-bladder, or spleen; the portal suppuration arising through an extension of this inflammation along the wall of the implicated branch, and so differing from embolic abscess of the liver in this, that the latter is due to the conveyance of the cause of suppuration by the blood-stream, and not by the vein-wall. Both kinds of suppuration, however, as we see, are generally evidently due to disease in the field of origin of the portal vessels. A fish-bone penetrating, or an abscess of the spleen reaching the branches of the portal vein, has been known to excite this suppuration. But it is sometimes very difficult to determine whether suppuration of the vein may not be primary, and the abscess in the field of origin of the vein secondary, and caused by it. Thus, in the case of suppuration of the rectum which we have mentioned, there were other submucous abscesses in the stomach; and it was very hard to say how these were related to the portal suppuration. Indeed, the abscesses in the rectum which we have described as reaching the veins might be believed to have themselves arisen by the extension of the suppuration down from the portal vein; it is rather by analogy with the frequent occurrence of phlebitis from evidently primary wound or ulceration near veins, that we adopted the belief that the abscess in the rectum was the cause of the portal abscess, rather than its effect.

In a few cases we have known very minute abscesses scattered here and there through the liver in the track of the portal vein, but really originating in the hepatic arteries. These were cases of ulcerative endocarditis, in which particles from the inflamed endocardium had entered these arteries, as well as the cerebral, splenic, and renal arteries, in the course of which vessels, throughout the organs which they severally supply, abscesses were also present. We must further notice, though it is perhaps too obvious to need remark, that all large abscesses of the liver, destroying as they do great areas of its tissue, must cut across more or less considerable branches of the portal vein, as, indeed, of the hepatic vein also. It is interesting to observe how the vein so cut across is preserved from the entry of pus. You will generally find that the suppuration does run some little distance along the hepatic great, so that one fifth or one third may be composed of fibre, and that even when the liver is above its normal weight. This fact proves the error of those who would deny that the fibrous substance of the liver augments in cirrhosis, describing the change as mere wasting of the cell-elements of the lobules, which, of course, would give only a relative and not an absolute excess of fibre. We have already seen in chronic atrophy that the wasting of the cells leads to no such result, and the enormous proportion of fibrous substance in an enlarged cirrhosed liver places the fact of its excess beyond doubt.

Hitherto we have spoken of this substance as fibrous tissue. Microscopic examination, however, shows in it elements responding to its active and encroaching nature; it is crowded more or less with small cells. It is often full of networks of biliary ducts, which have led to the supposition that in it an effort arises to restore the hepatic tissue again by the same outgrowth from the ducts as, you know, originally produced the hepatic tissue; further, it is loaded with large vessels, easily injected from the hepatic artery, for the compression exerted by the new formation tells more upon the feebler current in the portal vein, so that the hepatic artery enlarges to compensate.

The check to the portal-vein stream may in severe cases lead to entire stagnation of blood in that vessel, with coagulation; and after this has occurred, life may be prolonged at least many weeks, as the changes in the clot have proved. Under these circumstances, however, bile continues to be formed from the hepatic artery, and that even, apparently, in normal quantity, so that the fatal evils are those attendant on engorgement of the portal system,—ascites, wasting, hæmorrhages into the alimentary canal, &c. Once, indeed, we have seen rupture of the portal vein, leading to a large aneurismal sac which extended up behind the liver.

While the new fibro-cellular matter thus compresses the portal system, it perpetually invades the lobules, spreading between them, and passing into them, along the course of those considerable portal venules that enter them. Thus, it pierces and divides them up, isolating small portions and strangling the fragments, until only a few scattered cells remain.

The hepatic cells appear not to transform into the fibrous tissue, but to perish by simple atrophy, in the progress of which they become loaded with fat and bile-pigment, remains of which substances are seen in their places when all cellular form is lost. We thus have present not a simple loading of the liver-cells with fat, as in common fatty liver, in which the cells never break up, but a veritable fatty destruction of the cells.

We need scarcely say that cirrhosis of the liver is often found complicated with its other diseased states; especially with syphilitic changes, lardaceous disease, the nutmeg state in heart disease, and, as we have just now said, the various degrees of fatty change. In a pure case of cirrhosis the capsule of the liver is not thickened much, but some examples will be found coated with a more or less, and often very thick, capsule of adventitious tissue; this is always readily separable from the liver, leaving its proper capsule still on it. Such a coat exercises an immense compression upon the liver, flattening the gall-bladder and rounding the edges in a remarkable way, often by bending them over, so that when the coat is stripped off, the edge of the liver is found bent down upon the under surface of the organ, from which it rises when the false membrane is raised. A further proof of the tension exerted by this membrane is curiously given by its own appearance, which is always areolated, round holes extending more or less deeply into its substance. These are gaps which it has torn in itself during its contraction. When such a membrane exists upon the liver, one like it is generally found upon the spleen also. It is almost constantly a result of spirit drinking; indeed, it is one of the surest post-mortem evidences of that habit, and yet it is often present when there is no trace of cirrhosis; also, as we have said, advanced cirrhosis is often unaccompanied by it. A practical importance attaches to this thickening of the hepatic capsule, because of its liability as a chronic peritonitis to extend over the stomach, colon, and omentum, compressing and binding these parts together, and reaching the small intestines, to use them in the same way. When *paracentesis abdominis* is performed under these circumstances, chronic peritonitis is liable to become acute. The condition may be suspected when with moderate ascites the surface of the abdomen is dull to percussion.

It has been suggested as a possibility that cirrhosis of the liver sometimes commences by extension inwards of an inflammatory thickening of the capsule, such as we have described. In the case of the lung such an extension from a thickened pleura is believed to occur, so that chronic pleurisy leads to induration of the lung. That such a process outside the liver would cause a contraction and an impeded flow of blood through it sufficient to give rise to dropsy there can be no doubt; but as to whether it may actually involve the tissue to produce cirrhosis there is more uncertainty. It is just possible, however, that here may lie the explanation of those cases of cirrhosis which sometimes occur in young and temperate persons; that the affection has commenced as a simple peritoneal inflammation, and then the organ beneath has become involved, as you see in this drawing of a hobnail liver, taken from a girl eleven years of age, who died in this hospital a few years ago.

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In all cases, however, especially where more than one local fibrous scar-like patch exists, search should be made for other evidences of syphilitic taint in the body, and this with greater probability of success when the scars are on the surface and adherent to the adjacent parts. Multiple scars of this description almost always arise from syphilis.

Syphilitic Fibroid Deposits, or Nodules.—These deposits occur in the liver as well as elsewhere, in the same way as nodes appear on the bones, or as deposits of fibroid tissue in the muscles, or as similar deposits in the various other parenchymatous organs, the brain, spleen, testes, &c. The leading character of these changes is their strict localisation; the part affected undergoing extensive changes, while the remainder of the organ is healthy. Some authorities set down as the result of syphilis a universal induration, such as we have described to you under Cirrhosis, and they also admit a syphilitic form of capsular inflammation or *perihepatitis*. But if so caused, such changes could not be known from those due to spirit drinking, &c., whilst the latter cause would probably often coexist with the occasion of syphilis. It is very common to find lardaceous disease of the liver as well as of other parts, when the syphilitic change is extensive. The outline of the liver is altered more or less by the true syphilitic deposit. Scar-like depressions mark the surface; they may be so numerous and deep as to divide the liver up into a cluster of irregular masses, to which the name "botyroid" has been given, from its likeness to a bunch of grapes; or fixing especially on one lobe or part of a lobe, the disease may entirely destroy it; the remainder, which as we have said, is healthy, undergoing hypertrophy in compensation.

When a section is made through such a syphilitic scar the characteristic syphilitic gumma may be seen, but not always; for after an uncertain but usually long duration, the caseous gumma disappears, and nothing but fibrous tissue is left. The earlier stage of syphilitic action is rarely seen in the liver; it appears as a sharply localised, deeply reddened, swollen patch, the portal venules of which have a grey lymph around them. What we more usually see, however, is a later stage of the disease, when there is a pale yellowish, cheese-like nodule of irregular outline, surrounded by a fibrous zone, the outer edge of which disperses in the lobular tissue, the lobules dwindling gradually in its grasp. This fibrous zone is never very broad, the change is, as we have said, strictly localised. The cheesy centre varies in consistence from a gristle-like toughness to a pulpy softness; it is sometimes mortar-like from cretaceous change. We have known the centre to soften down and open into a bile-duct, but this occurrence is rare.

The size of the patches differs very much; sometimes they are as large as a good-sized plum or even occasionally greater; they range from this down to the size of a pea. We have never known this change of itself destroy life, for the remainder of the liver is healthy and hypertrophies. But in fatal cases of cirrhosis with lardaceous change, it often contributes a large and important share to the whole damage. As is often the case in pathology, the naked-eye appearance is far more distinctive than the microscopic characters, in which indeed there is nothing peculiar; the fibrous zone has much the appearance of severe cirrhosis, and the caseous nodule presents decaying fibrous and cellular elements, not differing from the inflammatory cells of ordinary granulations. No doubt a great number of what were formerly called "tubercles" were of this nature.

Albuminoid Infiltration.—This name has been given to a state of the liver presented by it, in common with the spleen and kidneys, in rickety children. The organ is paler, harder, and more elastic than natural. Close examination shows general increase of the connective tissue around the portal vessels, augmenting this to about four times the proper quantity. The microscope shows at the same time a corresponding but less considerable augmentation of the stroma in which the cells of the lobular structure are embedded. These characters correspond so far with the description of cirrhosis; but the organ is smooth in rickets.

Infantile Syphilis.—A very similar state of the liver is found in many children who have died of congenital syphilis. The disease in these cases frequently eludes observation, the hardness and watery bloodlessness of the tissue make its most evident macroscopic changes. The hardness and elasticity are so great that if small pieces are cut, and slipped with pressure under the finger they spring somewhat as though made of india-rubber.

There are, no doubt, other changes in the liver to be occasionally met with under various circumstances; thus, in case of poisoning by arsenic, when the liver has become affected through absorption of the poison, we have seen the organ present a peculiar appearance; in one, particularly, we remember it was pale like a fatty liver, and soft, with a pinkish hue in parts, and here and there an arborescent ecchymosis; this purpuric tendency in poisoning by arsenic we have already spoken of in its effect upon the heart.

Injury.—Rupture of the liver is very common; but, as it seldom occurs without a very severe fall or blow, forms often part only of an extensive fatal injury. As we before mentioned, the laceration of the abdominal organs mostly occurs by tearing, due to a stretching of the tissue; and thus, in the case of the liver, the most convex parts on its upper surface give way, although these are the most protected. If the injury be slight, the surface is fissured or cracked, just as can be artifically produced by violently bending the organ upon itself. If this be the only injury, and no great hæmorrhage occur, recovery may take place, for a laceration of the great organs in the body is by no means necessarily fatal. Thus, in a case which we examined some years ago, of a man who fell from a scaffold, injuring his head as well as his abdomen, and whose death occurred some weeks afterwards from cerebral disease, there was a long laceration in the liver quite healed; and as regards the blood which had escaped from it, this was seen to have produced a dark layer all over the peritoneum, a part having been absorbed, and a part remaining as a pigmental covering to the serous layer; there was, however, no inflammatory product. It is worthy of remark that in some of these cases large bile-ducts are torn through, and bile freely effused along with the blood, giving a deep yellow stain to the lacerated surface, and yet no inflammation is set up thereby. The torn surface of the liver is tolerant of its own secretion; even when the peritoneum was reached by this yellow stain, the fierce peritonitis which bile would be expected to produce certainly did not occur in any of our cases. Here are preparations and drawings, showing lacerations of the liver undergoing repair; and in a case we lately saw, where a bullet had penetrated the diaphragm and lodged in the liver, it appeared as if a cyst were about to form, to render its presence innocuous. When the liver is directly wounded, this is very often from fracture of the ribs; there is, of course, puncture of the diaphragm and thus often hæmorrhage into the chest. Here is a specimen showing a needle in the liver.

Spontaneous Laceration.—We have no well-authenticated case of spontaneous laceration; but it may be that such an accident has occurred, for it is remarkable how frequently we from time to time hear of instances of injuries to some of the abdominal organs arising from violent exertion. Thus we have had a case where a man made a violent effort to extricate himself from machinery, during which it appeared that he had ruptured the hepatic artery. At the same time we read of a girl who in like manner ruptured her gall-bladder. In this specimen of liver, in which you see several lacerations on the surface, it is said that death took place very speedily after violent vomiting; the inspection showed a large quantity of blood beneath the peritoneum which had proceeded from these rents in the organ. This reminds us that we have already shown you a stomach which was said to have been rent during vomiting. We mention these cases merely to state that there is some amount of evidence, though it is slight, towards showing the possibility of spontaneous lacerations of the liver. On this subject, however, it is very important to remark that where the liver has been ruptured either by blows or by being run over, no sign whatever of the injury may be found either on the surface of the body or in the abdominal parietes. So that in no case of reputed spontaneous rupture can post-mortem evidence afford any *proof of its spontaneity*.

Morbid Growths.—The liver is very subject to these, like the lung, but each organ shows preference for particular tumours.

Adenoma.—The liver is, according to our experience, rarely the seat of innocent or non-infectious tumours, while it is the favourite place for the formation of secondary cancerous growths of many kinds, especially of carcinoma proper. Of any tumours composed of true liver-substance developed in isolated incapsulated nodules, such as are described by Rindfleisch and others, we have little or no experience. In the case of the thyroid gland and supra-renal capsule such strictly *analogous* tumours are often met with, but in the liver we have never seen them. The accounts of the later development of these growths given by Rindfleisch bring before us a kind of tumour we think we have met with. But to call it adenoma is to employ that word in a sense which appears to us to be destructive of its usefulness as fitted to apply to such instances as the hypertrophic tumours of the breast, thyroid, &c., in which the natural structure is essentially preserved, whereas these so-called adenomas of the liver become widely different in structure from the hepatic substance.

Cancer.—The appearance of cancer of the liver varies much in different specimens, both according to the extent of development and the character of the disease. Sometimes the liver is of enormous weight, and yet only small patches of true liver tissue remain scattered through the substance. In such cases parts of the liver will occasionally be found in a very perfect nutmeg condition. This we have traced to an obstruction of the corresponding hepatic vein by pressure of the cancer-masses. The appearance of the cancer differs also in different cases, very much through this, that in some it forms bold tumours that push aside the tissue, while in other examples it alters the tissue without much displacing it, the lobular substance being swollen and changed to the colour of the cancer growth, but still retaining a great deal of its natural construction. This latter form is described as infiltration. The first kind, the expanding and compressing tumours, are usually rapidly growing *medullary carcinoma* or *sarcoma*. One may ascribe the reason of the circumferential compression to the circumstance that the multiplication of the elements within the affected area is greater than the rate of infection of surrounding tissue at the growing margin, so that the increasing interior mass pushes forcibly upon the as yet unimplicated surrounding liver. But in the infiltrating kind, which are generally the slower growing forms of scirrhus, often secondary to cancer of the breast, the spread of cancerous infection is more rapid than the increase in number of the cells in the infected area; hence there is no over growth and pressure, but the marginal increase keeps pace with the central, and the order of the infected parts is little disturbed. An infiltration, however, in a sense of a pouring in of cancer among the healthy tissues is not to be seen here or anywhere, for the cancer is not added to the tissue, but composed of the transformed tissue itself. Another feature of liver-cancer is the cupped appearance presented by the large nodules or masses that rise above the surface; this feature is the more common, because so large a part of hepatic cancer-growth is found at and involving the surface. Why this should be we cannot say; but if you do not see cancer on the surface of a liver, you are nearly sure not to see it on section. The cupped form of the cancer is due partly to the degenerative changes which its older central portion always undergoes, for however unfortunately obstinate the vitality of a cancer, this vitality is not due to any tenacity of life in its individual cells, which indeed are sufficiently frail but too prolific; hence the central parts of cancer waste, and contrast with the peripheral part, having the yellowish, bloodless, cheese-like appearance usually ascribed to tubercle, which appearance was indeed formerly supposed to signify mixture of tubercle with the cancer. These cheesy patches show microscopically decayed and perishing structure, but of the same nature as the rest of the cancer, whose *necrobiosis*, as it is termed, or more shortly as we will say it, whose *senescence* it indicates. Besides the natural senescence of the cancer cells, other influences work to produce the wasting and sinking change in the middle of the cancer; thus the growth of its elements is disorderly, and it penetrates its own vessels of supply, starving itself. This is especially true as regards the portal veins, through which indeed cancers of the liver cannot generally be injected; their supply of blood being kept up by the hepatic artery, through which, on the other hand, an injection of their living parts may be usually effected. Another cause of the cupped form of the superficial cancers arises thus, that the centre of the growth commenced when the cancer was small, whilst the margin of the growth grew in its later and always more active time, so that the latter is more voluminous than the former, as one sees in an oyster-shell, where the old part near the hinge is small and depressed.

The cancer, as we have said, is generally secondary. In the only two examples we have seen, in which the liver was the only organ affected, the cancer was a rather firm example of carcinoma. In the other cases, the secondary cancer, as usual, has the general character of the primary cancer, but is often softer and more rapid in its growth. The kinds of cancer that occur in the liver are chiefly *carcinoma, medullary sarcoma*, and *melano-sarcoma;* occasionally we have met with *cylinder-epithelial carcinoma*, and *colloid* may very rarely be met with also.

Carcinoma may be either of medullary softness or of scirrhous hardness; in the former case the cut surface yields, when scraped, a large quantity of characteristic creamy "cancer-juice." It has the usual brainlike character and softness, and does not infiltrate the tissue, but forms large thrusting masses, of which we have spoken. The softening which decay brings in the centre of these cancers is often extreme, so that they may appear like abscesses, and by this softening blood-vessels may be opened and hæmorrhage may occur into the soft space, which thus may become distended into a great blood cyst. Or smaller hæmorrhages of various dates, with the usual changes in the blood-pigment, may give different shades of brown and purple to the section. When these hæmorrhages occur a similar hæmorrhagic character is usually found in the primary cancer.

Another effect of the invasion of vessels by the cancer is of great interest; thus, approaching a large branch of portal or hepatic vein, it grows into and roughens the channel of the vessel; blood clot is deposited on this rough spot, and fills the vein. Cancer then *grows into the blood clot*, and so a large, soft, carcinomatous plug is found filling the vessel; such great soft cancerous clots have even been found without any implication of the vessels' wall. We have certainly seen such examples.

But another source of similar cancerous obstruction of the portal vein consists in *embolism*, as when cancer of the stomach enters and fills the gastric veins. In one remarkable case a continuous cancerous thrombus extended from the stomach cancer, through the gastric and splenic veins, and the whole length of the portal vein, into many of its smaller branches within the liver. This thrombus stuffed the vessels like sausages, but was only here and there attached to their wall.

Besides this extension within the veins, we occasionally meet with very hard cancer taking the place of Glisson's capsule around the portal veins, and also commonly surrounding the gall-bladder. Such growth is, however, generally rather sarcomatous or lympho-sarcomatous than carcinomatous in structure.

Scirrhus is often of the infiltrating kind. All varieties of consistence, from the medullary kind just mentioned to an almost cartilaginous hardness, are met with in cancer of the liver. The physical cause of the difference in consistence lies in the proportion which the fibrous network bears to the cellular contents of its interspaces. In scirrhous cancer, the cells are few and soon degenerate, so, indeed, that in the older central part little more than interlaced fibrous tissue may be found, with fatty relics of cells; while in the outer parts, of recent formation the cells are more numerous; the naked eye sees a reticulated or hyaline appearance.

From the section of these cancers, but little milky juice can be scraped, and yet this kind is often very infectious. We have generally seen it secondary to cancer of the breast.

Melanosis.—Wherever this exists to any extent in the body, the liver is liable to be affected, and thus we have several specimens showing the disease.

Colloid probably never occurs in the liver as a primary disease, but the organ is sometimes involved when the peritoneum is affected, as in the beautiful specimen we have already shown you.

Of late years much attention has been given to discover the mode of origin of cancer in the liver. From what we have seen we have no doubt that, as a rule, the cancer cells are developed by direct transformation of the hepatic cells. It has been asserted that the chief agents in the cancer-cell development are the epithelial cells lining the small blood-vessels. The excessive difficulty of these investigations, or rather the ease with which false conclusions are drawn, must throw doubt on the conclusion. But we can so far bear out this observation as having seen in one or two examples an extension of the cancer-growth between the hepatic cell-columns compressing these, and thus having the distribution of the capillary vessels.

Cysts.—Besides hydatids, you will find sometimes upon the surface of the liver small cysts, with very thick walls, as you see here; sometimes they may arise from *obstructed ducts*, thus in the case we just now showed you, where there was a chronic induration of a part of the liver from injury, a duct was obstructed, so that there was a large cavity filled with inspissated bile at the spot; and the same appears to be the cause of the cyst in this preparation, and also of the one seen in this drawing.

Cystic Disease is seen in this very remarkable specimen (see details in vol. vii, 'Path. Trans.'), associated with a similar cystic disease of the kidney. You see this portion of the liver, which is preserved, is full of cysts of all sizes, some as large as a walnut, and others smaller; they are separated by membranous partitions, and on careful examination of the ducts we cannot discover that they are in any way connected with them. Cruveilhier gives drawings of livers containing cysts, which he thinks depend on the dilatation of the ducts. Such may no doubt be sometimes the case, but the present specimen appears as a real cystic disease such as occurs in other organs.

Here is another specimen, containing very minute cavities or cysts, but whether it be a pathological condition we are not quite satisfied, for we should remind you that in very rapid decomposition, a similar appearance may be produced from the development of gas in the tissue; thus a liver some of you saw the other day resembled a mass of well-raised dough, and looked very much like this preparation.

Cavernous or Erectile Tissue.—We have several specimens on our shelves called blood tumours, supposed in some instances to have resulted from injury, but they are in reality distinct formations or growths of a cavernous tissue. In this specimen you see merely a red tumour, but in this other, from which the blood has been washed out, a very beautiful reticulated structure is left. Such formations are mostly met with on the anterior edge of the liver, where we have several times seen them; they are of about the size of a marble, and of a deep red colour. When cut across you see a cellular structure, the spaces being filled with blood, which comes from the portal vein, with which they communicate. Although they are called tumours, yet they do not project from the liver nor compress it; indeed, the affected part is often sunken slightly below the surface, upon which it is seen before section as a dark spot with an angular outline.

This fact, we think, must be considered in opposition to the opinion given by Virchow that these cavernous patches are essentially tumours with dilated venules. The amount of fibrous tissue in them is generally very slight; the network very delicate, and covered with a single layer of squamous epithelial cells of elongated form. They may be injected from the portal vein or from the hepatic artery. As to their importance, they are chiefly of scientific interest; they were formerly thought to represent an early stage of malignant growth, an idea which was easily reached when such growths were thought to be due to changes in extravasated blood. We have seen them associated with cancer on two occasions, but there was no sign of transformation of the cavernous tumour into cancer; they are not very unfrequent, and usually occur in healthy livers. The only change we have seen in them was a solidification of a part of one, evidently due to coagulation of the blood in it, as it presented all the characters of degenerating blood-clot.

Leukæmic Liver.—The liver in leukæmia is found generally very large, and its substance is diseased in a peculiar and characteristic way. Sometimes one can see on section minute grains resembling miliary tubercles, but soft, scattered in immense numbers in the course of the portal venules; generally, however, the distribution of the diseased change is so minute that it is not visible to the naked eye; a paleness and largeness of the lobules only being observed. Microscopic examination reveals collections of crowds of lymphoid cells, mostly following the course of the capillaries; these cells are very like those white cells whose excess in the blood constitutes leukæmia; differing from common white blood-cells, if at all, only in this, that the nuclei are larger and of bolder outline. The relation of these cells to the vessels has induced some to believe that they have wandered out through the vessels' wall, but as we have elsewhere said, this disease approaches in many ways to the character of a truly malignant and infectious disease of the blood, that is, a cancer of the blood.

Tubercle.—We have already seen that tubercles appear in many organs under two wholly different conditions correspondingly different in their forms. These are the primary local tuberculous nodule or mass which becomes caseous and softens, and the secondary shower of scattered minute grains familarly known as miliary tubercle. It is important to notice that the first kind, viz. primary and local tubercle, does not occur in the liver; at least, we have never seen it. The older descriptions of such tubercles probably refer to syphilitic disease or certain forms of sarcoma. But the liver is commonly affected with a fair share of miliary granulations, in cases of miliary tuberculosis. These tubercles form small greyish or yellowish granules in the tracks of the small portal venules, many usually reach the surface, and upon these may be seen little patches of chronic inflammatory lymph, sometimes making such livers look sprinkled with villosities. In cases of this kind the hepatic disorder forms a small and unimportant share of the whole disease. It is true the liver is usually swollen, but a great part of the swelling is due to congestion caused by the obstructed circulation which the coexisting tubercles in the lung have created.

Hydatid.—This is more common in the liver than in any other organ; a fact which tends to bear out the history already given, in which it was shown that the ova of the tapeworm are taken up by a portal vein from the intestinal canal, and are then developed in the liver into round or cystic animals. Hydatids are generally single in the human subject, and so differ from the multiple hydatid seen in the livers of the sheep and other creatures; more rarely, however, two or three of them are found together in the human liver.

The cyst may be of any dimension; it may grow as large as one's head, and contain several pints of fluid; or it may die when young and of very small size. Such dead hydatids are constantly being met with on the post-mortem table, in cases where their presence has never been suspected. The hyatid itself consists of sacs composed of a thin pearly white translucent membrane, which, when torn, curls back the reverse way to its natural curve. If the animal be large, its wall is of tolerable thickness, and is laminated in structure; but it is so tender that it will scarcely bear its own weight when lifted from its position. Unless some accident has happened to the creature, its wall is tensely full of a fluid like water, of very low specific gravity, seldom reaching more than 1010, and containing a little chloride of sodium; only the slightest degree of opacity is communicated to this fluid by boiling, so that is resembles pretty closely the cerebro-spinal fluid.

The animal generally contains a number of smaller ones of a similar kind, and of all sizes. The large parent sac is lodged within a hollow formed in the liver, but having no connection with it; thus the hydatid drops out when the hepatic *cyst* or containing cavity is opened; the latter is formed in the liver itself in the same way as the wall of an abscess, by a fibrous induration of the hepatic tissue around the hydatid. As the animal grows, so the hepatic tissue gives way, and becomes hardened to form a wall around it. When the hydatid is situated near the surface of the liver and protrudes, the hepatic cyst is protruded also, thus appearing as if it were formed around the hydatid by some independent process of its own, but this is really not so.

The hepatic cyst, under these circumstances, often becomes very hard or bony; that is, earthy matter is deposited in its walls. When found accidentally, hydatids are mostly dead, and then the parent hydatid has shrunken, together with the smaller ones within it; these, instead of floating in the fluid of the parent, are all then collapsed and bound tightly together, and as might be imagined, under the circumstances, a number of collapsed spheres being pressed together, they encircle one another so as to form many apparently crescentic membranes. In these cases the sac-like bodies have degenerated, and a quantity of fatty or putty-like matter is found amongst them, in which a quantity of concrete bile-pigment is not unfrequently present.

If the surface of the hydatid membrane be looked at beneath the microscope, it appears to be structureless, but in section it is seen to have a number of concentric linear markings indicating a lamellated structure; sometimes the sac is barren, and when open is found to contain a clear fluid and to have no animalcule-buds attached to it. When this is the case there is most disposition to recovery by simple tapping, such hydatids really representing abortive specimens. More frequently the larger sac, and perhaps the secondary sac also, will have on their inner surface crowds of minute bodies looking like white grains. The inner surface itself is composed of a delicate translucent membrane in which these grains arise. Under the microscope they resolve themselves into clusters of exceedingly small round bodies, each cluster being enclosed in a delicate membrane, and attached by a stalk, which gradually narrows until they separate and become free. The minute round bodies which compose the cluster are found to show the anatomical characters of the head of a tapeworm. That is, they are armed with a close double circlet of well-formed hooks, outside which are four oval suckers at regular intervals a little distance off. The circlet and suckers, with the head which carries them, may be seen protruded in some of the examples from the little sac with which the head is continuous, but more often the head is retracted in an inverted state within the saccular neck, like the top of a glove-finger pushed in. Numerous grains of lime salts mark the surface of this sac. If the hydatid be decayed, the hooklets may, nevertheless, be found, since from their stony nature they are but little perishable. These heads are the young *scolices* of the *Tænia echinococcus*, a small tapeworm of not more than three joints, found usually in dogs. Long before their real nature was known, these heads were recognised by Dr Bright, as you will see them figured, though very vaguely, in the first volume of our 'Hospital Reports.' We have already said that the tapeworms form the mature state of the animals, but we have never yet found them at present in the same subject as contained the hydatid, and this agrees with the observation of experimenters, that the worm undergoes its stages of development in different bodies.

We have once met with a case in which an hydatid sac *budded from its outer surface*, and this process repeated itself so as to form a great chain of hydatid sacs, which extended through the diaphragm, and permeated nearly the whole of the lung without creating any inflammation of its texture. We have already mentioned the occurrence of a similar form of hydatid budding from the outside, in the bones, and elsewhere (see *Spine*). Neither of these two examples had any scolices with hooklets present; all stages of the formation, from the budding to the completion of a double cyst, and the repetition of the budding, could be seen; and thus the whole hydatid disease formed a chain of cysts of the kind called multilocular, occupied by the more or less mutually connected hydatids, the connection usually having broken down.

Several German authors have described a minute multilocular hydatid disease, extending in the course of the portal vein through the liver, and producing symptoms of obstruction of the ducts with fatal jaundice, but we have not met with any specimen of this.

The most usual termination of an hydatid is to cease to grow, die, and shrivel up. It may be killed by the entry of bile into its cavity. Or the fibrous encasing cysts of compressed and altered liver tissue may calcify; this, however, is more likely to be a consequence of slowness of growth in the parasite than the cause of its death. The opening of a *small* duct by the hydatid so as to poison him with bile is good for the host, but it is very unfortunate if a large duct be opened, and the hydatids attempt to pass that way; great pain, jaundice, and death, are the almost constant result; hydatids sometimes broach the vessels, and so cause hæmorrhage into their interior. If they continue to grow, their tendency is ultimately to burst, and make their escape. They may be broken by an accidental blow, a very fatal occurrence; or they may make their way into the pleuræ, or pericardium, stomach, colon, or into the vena cava, or outwardly through the skin. The risk of either of these occurrences is proportioned pretty closely to the extent of liver surface in contact with these several parts, so that, as would be expected, the right pleura against which through the diaphragm the liver so extensively abuts receives the largest share; commonly the lung becomes adherent, so that the hydatids are discharged through that organ, and may be spat up; this is again a very dangerous event. The most favourable direction is that into the alimentary canal, and many recoveries are recorded of cases wherein hydatids took this course.

DISEASES OF THE GALL-BLADDER AND DUCTS

Atrophy.—The gall-bladder is sometimes absent; thus in one case we found the gall-bladder represented by a shrivelled relic of fibrous tissue; death having occurred from painless obstruction of the ductus communis by a large gall-stone. In another remarkable case of extreme obesity the gall-bladder was coated thickly with fat which had so impeded its function that it contained only a little clear mucus, while the rest of the biliary system was full of bile. Its size varies in many forms of disease; where little food had been taken, or in actual starvation, it is found very full of bile. In an example like this where it is much distended, an obstruction existed to the discharge of its contents into the intestine; and such a case occurred here a few years ago, where the gall-bladder held more than a pint of fluid. This condition is called *hydrops cystis fellicæ;* but it is rather an inflammation than a dropsy. Cases are recorded where, from injury and subsequent fistula, the bladder has discharged itself externally, and bile has been poured out to the amount of eight oz. daily for a period of several months.

Dilatation.—We not unfrequently find the gall-bladder of large size when there it no evident obstruction to its duct; the enlargement being evidently chronic, as shown by a deep notch which its pressure has caused in the liver. We have never seen any evil results from such simple dilatation, though on one occasion five oz. of bile were present in such a dilated gall-bladder, without any jaundice. But the occurrence is not without interest in connection with similar dilatations of the stomach, urinary bladder, heart, ureters, &c., without evident mechanical cause. It appears that any hollow organ may slowly yield through inability to expel its contents, but it is always more satisfactory to find a tangible obstruction, which should be carefully searched for.

The most common cause of dilatation of the gall-bladder is a closure of the hepatic or common duct, by cancer or by a gall-stone, in fatal cases of jaundice. Behind the obstruction the ducts become enormously widened, so that they may be seen projecting from the surface of the liver; and they appear when opened as large as one's fingers, very frequently pouring out fluid to the amount of half a pint. When the dilatation is extreme this fluid is not bile, for the secretion at length becomes so impeded (the bile which previously existed having again been taken up into the blood), that nothing is left in the ducts but a thin mucus often perfectly colourless.

We think this fact has not been sufficiently recognised in the common simple theory of obstructive jaundice, which supposes that the bile, impeded in its flow into the duodenum, is taken up into the circulation through the lymphatics and vessels of the walls of the gall-ducts, thus causing the general yellow colour.

Now when we find the body most deeply jaundiced, and the blood-serum dark orange-yellow with bile-pigment, it is surprising to discover that the gall-ducts, which are supposed to be the source of the bile furnished to the blood, are themselves devoid of bile and contain a colourless fluid. This indeed proves that the gall-ducts are not the source of the bile, and that the bile in the blood in these obstruction cases is absorbed from the liver tissue, if it be absorbed at all. The question is thus raised whether it is not by suppression of secretion that the jaundice is caused here as well as in the unobstructive jaundice of yellow atrophy, fevers, &c., as, indeed, we are disposed to believe. The clear white slightly viscid fluid present in the ducts under the circumstances is a thin mucus secreted by the walls of the ducts in which, as you know, numerous mucous glands are embedded. There is commonly no albumen in the fluid, and even by the nitric-acid test no colouring matter of bile can be discovered. You will see in this specimen how enormously these ducts become dilated; the liver is then of a dark green colour, and it was stated by Dr Thomas Williams some years ago, in our 'Hospital Reports,' that the secreting cells became destroyed; and no doubt there is an important truth in this statement, although the liver cells are not altogether broken down, but rather shrivelled and defaced, and pigmented, and only here and there quite disintegrated. In some cases, however, the destruction of cells is very marked; the microscopic condition approaching that found in yellow atrophy. It is here important to notice that the fatal termination of such cases of obstructive jaundice not unfrequently takes the form of apoplectic coma from blood poisoning; the attack may be sudden, but no blood is found; death being due to bile poisoning as in yellow atrophy.

Acute Inflammation of the ducts has sometimes arisen from injury, and has occurred in cases of general suppuration of the liver which has involved these parts.

We have met with a few cases of acute idiopathic inflammation, the gall-bladder being thick walled and reddened, and containing mucus. Sometimes a layer of mucus adhering to the wall whilst the bile occupies the centre. As an idiopathic affection, this is extremely rare. We have found it in some cases of acute yellow atrophy of the liver, and on other occasions it has been associated with gall-stone, probably as a result of irritation by these concretions. Such inflammations of the gall-bladder with formation of pus in the wall, or great infiltration of it with lymph, or ulceration of it, have been met with in fevers and in cholera.

Catarrh of the bile ducts is a term which, as you know, is frequently employed at the bedside to explain the occurrence of simple painless jaundice. As these cases all recover when uncomplicated, they may be said to have no morbid anatomy; so that it is only as a matter of speculation that inflammatory mucus or turgescence of the mucous membrane is assumed to exist.

You can easily prove by experiment that no pressure of the gall-bladder through the parietes could exert any important pressure upon the ducts; so that no good could be expected from this proposed plan of treatment; but the gall-bladder, if thin or ulcerated, might be burst, an accident which has occurred during such attempts.

Chronic Inflammation and Ulceration.—These states are nearly always due to gall-stones, or at least accompany them. But a few cases of simple stricture are on record. The changes which gall-stones produce in the gall-bladder and on parts around make one of the most intricate and important chapters in pathology. The simplest condition we have observed, as a result of gall-stone, is a widening and slight thickening of the whole length of the duct from the gall-bladder to the duodenum; this wide duct having several gall-stones in it. We have twice known this occur when relatives of the person have denied the presence of pain or other biliary symptoms during life. Indeed, gall-stones are not unfrequently latent; this is, however, usually not because they pass without symptoms, but because they do not pass out of the gall-bladder. It is not very uncommon to find a large gall-stone firmly impacted in the neck of the gall-bladder, entirely closing it. The bile in the gall-bladder is then absorbed, and replaced by a thin mucus; and the walls of the gall-bladder are usually more or less thickened. Sometimes gall-stones produce severe inflammation of the gall-bladder, causing it to adhere to parts around, usually the duodenum, and by ulceration making a perforation by which the gall-stones are discharged. In this way very large gall-stones may be got rid of, but many risks of fatal danger beset this kind of relief. If the stone is large it may block up the small intestine and kill by obstruction; this end is relatively not very unfrequent; or the ulceration may set up pyæmic abscess of the liver, or sometimes suppuration extends by continuity along the ducts, in the tracts of the portal vein. Sometimes the obstructed gall-bladder suppurates and forms a large abscess. It occasionally happens that the gall-stones are discharged through the abdominal wall; this may occur without any previous jaundice, and so the case be mistaken for carbuncle, &c. The real nature of the case being further obscured by the course of a sinus for some distance through the abdominal wall so as not to point over the gall-bladder.

More rarely the gall-bladder opens into the colon. In two such examples we recently met with, the gall-bladder was the seat of extensive cancer, and this is usually the case when the colon is opened by it.

In yet other examples the gall-bladder opens, commonly by rupture, into the peritoneal cavity, and, again, in very rare instances gall-stones have been passed in the urine. It is almost doubtful whether the gall-bladder ever communicates directly with the stomach in this manner, though the opening may be close to the pylorus instead of about the second flexure of the duodenum.

Gall-stones, however, are occasionally, but very rarely, ejected by vomiting. One was shown us the other day by Mr Mann, of Peckham. It had been vomited by a patient who had long suffered from chronic jaundice and hepatic colic.

Morbid Growths.—Cancer in these parts is not very unfrequent. We have met with several cases where a small knot of cancer has grown in the bile-duct wall, and proved fatal by a very chronic painless jaundice. Once the hepatic duct alone was affected at a point not half an inch in any diameter, the substance of the formation resembling the local hypertrophy of gland-tissue in the mucous membrane of the mouth we sometimes meet with. In this case, as in other very prolonged obstructive cases, *xanthelasma* was present. A more common seat of small tumours obstructing the duct is about the *ductus communis*, near the head of the pancreas. We have twice known syphilitic gumma form in this region, and cause jaundice by its pressure on the duct; once it softened and burst into the duodenum, and so the jaundice was relieved.

In other examples the new growth extends along the course of the portal vein, appearing as a whitish thickening of Glisson's capsule, and perhaps extending to and involving the gall-bladder. Some instances of this, offer a not uncommon difficulty in distinguishing between inflammatory thickening and tumour, the substance following the course of the ducts, and being of an almost purely fibrous structure, with scarcely any cell-element. One that was recently shown at the Pathological Society was composed of lymph-gland-like substance, and the disease was associated with a similar change in the wall of the duodenum.

We recently met with a curious example of disease. The common duct in a child of four years was dilated to form a cyst as large as the child's head. In the wall of this cyst were crowds of pendulous growths of myxomatous structure, in which were embedded numbers of non-striated muscle fibres. The gall-bladder itself is sometimes primarily affected with cancer, which may enlarge it enormously; it then usually contains gall-stones, and it may, as we have said, open into the colon and discharge these. A consideration of the history of such cases leads sometimes to a belief that the cancer follows upon the gall-stones as a result of irritation of the gall-bladder through their presence. Twice we have seen this cancer take the villous form, the cavity being enormously distended, but occupied by crowded dentritic growths. The liver may be invaded by contiguity of such cancers, or secondary growths may arise in it.

Ossification, fatty degeneration, &c.—In cases of xanthelasma from chronic obstructive jaundice, where the ducts were greatly dilated, we have seen the lining of their walls in a state of fatty degeneration similar to that which produces the patches in the skin. Occasionally we find a calcareous degeneration of the gall-bladder and ducts, the inner surface of these being covered with white deposits of carbonate of lime; this seldom reaches to any extent, or approaches to what is seen in the liver of the sheep or of the ox. Thus, in this specimen from the latter animal you see the ducts forming rigid long tubes, and within them are these flukes (distoma hepaticum); but whether the parasites are the cause of the disease we cannot say. In speaking of parasites it has been stated that the ascaris lumbricoides has occasionally penetrated the gall-ducts; and the proof that the worm had reached there before death was the fact of its having caused an abscess around the part where it was located.

Gall-stones.—The usual seat of these is the gall-bladder, as you would expect, or else some point in the course from the gall-bladder down the ducts to the intestine, as we have already seen; but now and then biliary concretions are found in the hepatic ducts within the liver. They are then generally quite minute, constituting what has been called biliary gravel; but sometimes, as in this instance, you see large calculi in the ducts of the liver itself. The cause of the production of biliary calculi has never yet been clearly ascertained; they are constantly met with when the liver is quite healthy, and this we also know from the fact of many persons being in perfect health after passing them. They may, however, coexist with cancer, cirrhosis, and other diseases. The tendency to their production increases with age. They consist of elements of the bile, and we can easily imagine how these might form concretions from some change or decomposition of that fluid, whether this occurred spontaneously or from disease of the coats of the gall-bladder. The constituents of gall-stones are cholesterine, bile-pigment, and earthy matter; the last two combine in a definite compound called pigment-lime. Some calculi are formed of pure cholesterine, and then constitute very beautiful translucent crystalline bodies. In other calculi this substance may be seen as sparkling pearly plates, or, if not visible, may be procured by boiling a piece of the stone in alcohol, and pouring the solution on a slide, when the crystals readily form; they are easily recognised under the microscope as rhomboid plates, lying one over the other, and with one angle often deficient, the two perfect angles at either end being 100° and 80°.

Most gall-stones, if of any size, are composed of earthy matter, pigment, and cholesterine; and if one be split across it is seen of a brownish colour, and formed often of concentric layers, with pearly plates glittering amongst them; the centre is often black, as if of pure pigment, and from this some radiating lines may be seen. A combination of all these substances is the most common, but sometimes the calculi are black, and of rough irregular surface, as if composed of pure pigmentary deposits; at other times of a white colour. Sometimes you see the gall-bladder full, as in these examples, and the calculi polygonal, from the attrition of one against the other; or rather, we believe, in many instances, simply from their compression against one another during their increase. More rarely they are found in the ducts of the liver, as we have already shown you, but if so, they may put on this irregular branching shape. As regards the cause of their formation, we know nothing positively, but we think it can be easily seen how very readily they might be produced. We have said that the cholesterine, pigment, or biliverdin, which constitute calculi, are also ingredients of the bile, and therefore it can be readily perceived how, if once a nucleus existed, a precipitation and concretion would result in the same way as the urinary ingredients form a calculus around any foreign body in the bladder; and if there should be no such cause, we can see how a change in the constitution of the bile might lead to a precipitate, for, besides the substances already mentioned which exist in bile, there is another, its principal constituent, a resinous substance, or the *bilin* of Berzelius, which is now known to be a compound salt, called the cholate of soda (glycocholate and taurocholate); if some of this salt be added to a gall-stone it will cause its solution, whence it can easily be understood how any cause operating, either in the liver or the walls of the gall-bladder, to alter the character of the secretion, and destroy or lessen the amount of this peculiar principle, would tend to the precipitation of the other ingredients; but whether such method is in operation for the production of gall-stones we do not know.

DISEASES OF THE PANCREAS

Diseases of the pancreas are not very common; we mean those diseases which are connected with great structural changes; that is, such as are so frequently met with in the lungs and elsewhere. The pancreas shares this immunity with the salivary glands, to which it is allied very closely in structure and function. It is very probable, however, that more minute alterations in the secreting structure may be constantly occurring during life, and that these are productive of symptoms in the same way as variations in the secreting tissues of the stomach, &c.

Inflammation.—*Acute.*—Although it is probable that changes may occur in the secreting tissue more or less allied to those called inflammatory in other organs, yet the more marked effects of inflammation, such as suppuration, &c., are not often seen. We have met with abscess on more than one occasion around the pancreas, and involving it, but it was probably only secondary; thus, we have met with suppuration of the pancreas in the later stages of typhoid fever. The other salivary glands likewise occasionally suppurate after fevers, pus forming about the gland as well as within it.

We have also specimens on our shelves which are described as *gangrene* of this organ. As you may sometimes find the pancreas involved in any suppuration which may be going on near it, so you may find the remains of inflammatory products within it. We have more than once found the pancreas occupied, as you here observe, with white amorphous matter, the remains of pus or other unorganisable exudation; in one case this was connected with a perforating ulcer of the duodenum which was undergoing cure.

Chronic inflammation and induration.—Chronic inflammatory changes of the pancreas are described by some authors; the anatomical evidence of such occurrence is not very satisfactory. But the pancreas is found to vary frequently in size and hardness; the clinical importance of such changes has also yet to be proved.

The most important effect ascribed to this induration of the pancreas used to be a compression of the common bile duct when it joins the pancreatic duct, and the consequent production of prolonged deep jaundice and wasting. We have occasionally seen cases in which the pancreas shared in a chronic thickening of the tissues about the bile-ducts, and generally of Glisson's capsule. The jaundice which accompanied these cases was due to the constriction of the biliary canals by the chronic fibrous formation around them, and not to the pressure of the indurated pancreas. The impeded function of this organ, however, doubtless aggravates the symptoms.

The pancreas is apt to be involved in ulceration of the stomach (this organ, you know, is placed behind the lesser curvature of the stomach, as the latter curves round the spigelian lobe), and thus it sometimes forms the floor of a large gastric ulcer, while the ulcer in spreading opens either the splenic or one of the pancreatic arteries, causing speedy death by hæmorrhage.

Morbid Growths.—We may here mention the occasional occurrence of accessory pancreas; one or more of these may be found forming gradations between the pancreas and Brunner's glands, which resemble each other very closely in structure.

Cancer.—No part of the body is free from a liability to cancer. This proposition is especially true if, by cancer, we mean tumour which is malignant in the sense of causing death—a latitude of signification too frequently allowed to this most unruly word. We use the term cancer only for malignant, that is, auto-infectious, tumours; but as we have seen elsewhere, tumours in these vital parts, although of an infectious nature, yet may destroy life before secondary growths have had time to arise; hence it is only by careful study of the structure of these tumours, comparing their anatomy with the different kinds of tumour found elsewhere, that we can decide upon those proper characters which their premature mortality nipped in the bud.

No doubt it is very wrong to speak of the chronic inflammatory indurations we recently referred to as *scirrhous*, and to confuse them with malignant disease; but other tumours occur which have all the characters of true carcinoma, ranging in feature from the soft and encephaloid to the hard contractile scirrhous form. Particular interest attaches to those cases in which a small knot of true scirrhus arises in or near the head of the pancreas, and drags upon, compresses, or otherwise closes the bile duct, and perhaps the pancreatic duct also, leading to tedious but generally painless jaundice and wasting. The pancreas, like the other salivary glands, has many lymphatic glands around it; and again, its substance is not enclosed in a capsule defined by a sheath of peritoneum, so that, as we have seen to be the case with the other salivary glands, it is often difficult to decide whether a tumour partly involving the pancreas, did or did not originate in its proper s stance.

The difficulty is, of course, much increased by this fact that glands are almost invariably implicated through the malignity of growth; indeed, it is often impossible to say whether the disease be in the pancreas or in the glands about its head. It is certain these glands do become involved in cancer whilst the pancreas escap and this has induced in some the belief that the pancreas nearly alw gets its disease by extension from the glands. Nevertheless, we h met a few cases where the disease was limited to the pancreas; form of the cancer will be of service in the decision of this questi for primary cancer of the lymphatic glands is almost always *lympho* while primary cancer of the pancreas is *carcinoma*. We have ne met, as yet, with tumours of the pancreas resembling the myxoch droma so common in connection with the parotid gland.

We have known the pancreas to have been affected with a *coll* form of cancer; thus, in this example, nearly the whole of it was volved, the only other disease in the body being a similar affection the omentum; death was slow and accompanied by excessive en ciation.

Tubercle.—We have never seen an instance of tubercle in the p creas; both it and the salivary glands appear to enjoy an immunity fr this form of disease. Occasionally, the remains of chronic abscess the form of more or less cheesy dried pus give an appearance such might be called scrofulous. We have before mentioned a case syphilitic deposit in the cellular tissue near the head of the pancre We have never seen that kind of disease within the organ; its occ rence there must be extremely unusual. We know of but one record instance.

Obstruction and Dilatation of Ducts.—We have already m tioned that such conditions are very common. In cases of constr tion of the common bile-duct in jaundice, the pancreatic duct may a be involved, and then, just as the hepatic ducts are dilated behi the stricture, so is the pancreatic duct widened, as you see in t specimen.

Occasionally cysts are formed in the pancreas, as this specim shows you; one we met with not long ago was as large as an e and contained a white, viscid fluid, corresponding to the pancrea juice.

Calculi.—As these may form in the salivary glands, so they m sometimes in the pancreas; they are generally small and multip These, which were analysed by Dr Golding Bird, consist of oxalate lime, phosphate of lime, and phosphate of magnesia. Those that fo in the salivary glands generally contain much carbonate of lime, and effervesce freely on the addition of acids.

Injury.—The sheltered position which the pancreas occupies places it almost out of reach, except by gunshot or penetrating wounds; but in this example, which was taken from the body of an adult who had been run over in the street, the pancreas is so crushed in its middle as to be divided into two parts. The injury affects the part opposite the spinal column, whose prominence no doubt afforded the counter pressure in the crushing action. There was but little other sign of injury to the abdominal viscera.

of the spleen is most intimately related to the blood which it contains. Some anatomists have described the arteries as opening into spongy splenic tissue, and the veins as emerging from this. Others suppose the pulp to be within the blood-vessels; and yet others, including the chief authorities, agree with Kölliker in the belief that a part of the blood traverses the pulp on its way from artery to vein, just as the lymph permeates its glands, while another part of the blood passes through capillaries in the ordinary way. We give these details because they must be understood before one is at all prepared for the exceedingly variable size and consistence of the spleen. The spleen, indeed, is a most sensitive index of blood changes, and is able to swell very quickly to a large size, as well as equally quickly to return to its normal limits. The mechanism of these changes is not fully understood; but when we apprehend the anatomy of the spleen, and perceive that it is a greatly expansible cellulosity into which the blood has direct and free access by the arteries, we can very easily anticipate that any hepatic obstruction, nay, that any obstruction which should render the flow of blood in the body importantly more difficult, would cause swelling of an organ so ready to yield under blood pressure. The speedy variations of size which the spleen undergoes are rendered possible by the elastic coat of the organ, which favours its return from the distended to the normal condition. The presence of this coat shows a provision for recovery from distension, and, as in the case of the lung, infers that the organ is intended to undergo such changes. In some animals, the recovery of the normal size of the organ is not left to its elasticity, but muscular fibres are introduced into the trabeculæ, and then a more active contraction of the spleen is provided.

In ague the spleen is large, and the shrunken, bloodless state of the surface of the body in the cold stage of the ague fit is, no doubt, partially due to the retention of blood in the spleen. Whether the retirement of the blood from the general surface causes the congestion and enlargement of the spleen, is more doubtful, and this doubt must be allowed to rest on all explanations of great enlargement of the spleen, which suppose it to be due to mere mechanical obstruction to the exit of its blood.

Moderate swelling, no doubt, occurs from such obstruction, and when the obstruction is complete, as in thrombosis of the portal vein, the size of the organ may be very great, say twenty-four ounces or more. But in ordinary cases of cirrhosis of the liver, although the spleen is often large, weighing generally about ten ounces, yet it may be small; thus frequently we have found it only five ounces in weight, and once it weighed only two and a half ounces in a case where the liver was covered with a thick capsule, and reduced to thirty-two ounces, its atrophy causing fatal hepatic ascites. Again, as we shall presently more fully show, the spleen in obstructive heart disease is usually small. These facts prove that mechanical congestion of the spleen is not the chief cause of its hypertrophy, and we shall find reason to believe that all considerable hypertrophies of the spleen are due to diseases, either of its own proper texture, or of the blood.

The colour of the spleen varies a good deal, corresponding to the several shades of venous blood; relieved, of course, by the presence of the pale coloured trabeculæ, and other white matters intermingled with the blood colour. As a general rule, the hue is not a consideration of much importance, except in the case of melanæmia, which we shall presently describe.

The colour of a section is always comparatively dark when fresh made; but exposure to the air causes oxidation of the blood, and thus from a dark venous tint, the cut surface changes to a light colour, approaching that of arterial blood.

Hypertrophy.—Mere accumulation of blood in the veins of the spleen will not account for most cases of its enlargement; the spleen not only enlarges but frequently also keeps its proper consistence. By hypertrophy of the spleen we mean a condition in which its tissue is simply increased with no alteration of its structure. The spleen is very remarkable for the large size it attains to, while still showing its usual characters in point of consistence, texture, colour, &c. Hypertrophied spleens are sometimes softer or harder than the average; these variations of consistence they, however, share with the normal sized spleen, which is very liable to alterations of consistence. We have remarked upon the very rapid increase of size which the spleen shows in agues. It is easy to suppose that if the enlargement from such causes were long continued the tissue might lose its elasticity and the organ would retain permanently its increase of size; this is a probable explanation of some forms of hypertrophy of the spleen; but while accepting such simple explanations we must notice what we have already mentioned, namely, that in heart disease, even when the liver is in a state of advanced nutmeg change from chronic engorgement, the spleen is often even smaller than natural; and the same is true also frequently in cirrhosis of the liver, for the spleen is not constantly enlarged in cases of cirrhosis, though it generally is so. But on mechanical grounds the spleen would be expected to be of great size in these diseases. Nevertheless, such facts must not induce us to deny that obstruction of the portal circulation will cause great increase of the size of the spleen, for in those not very unfrequent cases in which the blood in the portal vein coagulates ante mortem, the spleen is almost invariably very large.

The more common cases of considerable hypertrophy of the spleen are, then, not due to mechanical causes. If we should point to any class of affections that specially produce such hypertrophy, it would be to those chronic febrile states which directly implicate the blood; especially we would mention endocarditis. We do not mean by this a chronic mechanical obstruction of the valves, but processes of active ulcerative or other inflammation, which occur about the valves of the heart. Such endocarditis is a frequent cause of hypertrophy of the spleen; this may reach to from 16 to 30 oz. weight. Thus, the average weight of the spleen in ten cases of ulcerative endocarditis occurring in 1867-70, was 25 oz.; they were always more or less soft and pulpy. Very rarely the spleen reaches even to double the latter weight in endocarditis.

On the other hand, the spleen is usually quite small, as well as hard and dark coloured, in cases of chronic cardiac obstruction due to narrowing of the orifices or dilatation of the cavities of the heart; that is, in cases where *ischæmia* or obstruction to the course of blood is present, without poisoning of its current with inflammatory products. The spleen under these circumstances will generally not weigh more than from 4 to 8 oz.

In syphilis, again, we often meet with hypertrophic enlargement of the spleen. We do not now refer to the enlargement brought about by lardaceous change. In syphilitic bodies quite free from lardaceous disease, the spleen is often of large size: thus, in eight such syphilitic subjects, the average weight of the spleen was 19 oz.; no cirrhosis or lardaceous disease being present in them.

Prolonged endurance of tropical miasm is another cause of hypertrophy of the spleen; the enlargement results even when there have been but slight manifestations of intermittent fever; and this fact again throws doubt on the simple explanation of the hypertrophy as due to mechanical engorgement, and indicates that there is some special disorder of the relation between spleen and blood in these fevers.

We must not say that all chronic febrile states cause enlargement of the spleen; thus, phthisis does not do so, neither do the fevers that accompany chronic surgical suppuration, unless the lardaceous change intervene. Indeed, we think that when the spleen is greatly enlarged in any fever, this fact has a signification beyond that of the pyrexia or hotness, and indicates that there is a special disorder of the blood in that fever. The connection of the function of the spleen with the process of blood-formation is certain, although its nature is still obscure; and the state of the spleen may, we think, be considered in the light of a very valuable index of the state of the blood itself. The fever spleen is generally not more than from 8 to 15 oz. weight. The spleen in general tuberculosis is not more than 8 to 12 oz. In both these cases it is soft as well as large.

Enlargement of the spleen from such causes as agues, endocarditis, and fevers, however, merges more or less in the history of the primary disorders, although in some cases the disease of the spleen may be an important part of the whole trouble. Thus, in heart disease the organ may reach 3½ lb. weight, and this enlargement may be accompanied by capsulitis, and so may give rise to a grave addition to the whole suffering and danger.

But besides these cases there are others in which the spleen is enlarged to a great size, and this enlargement is the chief, if not the only disease that can be discovered. Thus, Mr Spencer Wells removed a spleen weighing 6 lb., this being the only disease discoverable in the body, and the blood not being leukæmic; and Mr Squire met with a spleen weighing 13 lb., while the blood did not show a positive excess of white corpuscles. We have met with similar cases of simple hypertrophy of the spleen without leukæmia both in the living and in dead subjects. They are generally, but not always, at least during life, associated with anæmia, and so, indeed, as a rule, are all long-standing diseases of the spleen. These cases are the only ones that can be properly considered as pure examples of hypertrophy of the spleen; the cause of the enlargement is unknown, and the accompanying clinical phenomena need further investigation.

We are not now speaking of Hodgkin's disease, or anæmia lymphatica; for in the cases at present under consideration the splenic tissue is normal throughout; whereas in Hodgkin's disease isolated patches of peculiar deposit are constantly present in the spleen. In the simple hypertrophies we are speaking of it is reasonable to suppose that the formation of white corpuscles is rendered imperfect, and they do not become free, but accumulate in the splenic pulp. It may be that such cases stand to leukæmia in this relation, that they represent a variety of it, in which the white corpuscles not only are unable to develop naturally into red corpuscles, but are even unable to free themselves from their place of origin and escape into the blood; while in leukæmia, on the other hand, the white cells can detach themselves from their birthplace and float off, but are unable to undergo their natural development into red blood-cells. This, however, is rather a mode of provisionally putting the facts together than a view of their nature. The spleen has sometimes been found enlarged in exophthalmic goître, but in the cases we have seen it was of natural size; once it was 4½ oz., or less than the usual weight.

Leukæmia.—In leukæmia the spleen reaches an enormous size. Thus one which Mr Bryant removed weighed 168 ounces, and this is not an extreme weight for the spleen in leukæmia. The tissue of the organ is generally not discoverably diseased, in spite of the great increase of size; indeed, the liver often shows much more of morbid change than the spleen in leukæmia. The liver in the above-mentioned case weighed 138 ounces. The tissue of the spleen in this disease varies somewhat in consistence, like ordinary spleens. Thus it may be rather flabby, or it may be firm and resistant. Its capsule is not generally inflamed or otherwise altered, so that any adhesions or thickenings of it must be regarded as accidental. The surface may present a minutely mottled appearance from fine white lines and dots forming a network in it. The same mottled appearance may be seen on the section, the natural deep purple colour of the splenic pulp being the prevalent colour, but this being mingled with the pale lines, which are sections of the trabeculæ and vessels of the spleen, and with the pale Malpighian corpuscles, which are often somewhat enlarged; in short, the usual appearance of the spleen, but coarser in its markings. The only microscopic peculiarity we have been able to discover is the filling of the small vessels with white blood-cells; but this appearance is of course universal in the tissues. We have several times met, in leukæmia, with *embolic infarctions* in the form of pale, circumscribed, wedge-shaped patches, with a deep zone of congestion round them; both the spleens removed by Mr Bryant showed these infarctions, although the general health appeared good when the operation was undertaken. In one of these cases there were similar infarctions in the lung, having much the appearance of pneumonic patches found in pyæmia, except that they showed no signs of breaking down.

The blood in leukæmia contains a very large quantity of white corpuscles, so large a quantity indeed that its appearance is much changed, and it has a creamy purplish look, which is very characteristic. This whitishness of the colour is well denoted by the name *leukæmia*, while lesser degrees of increase of the white cells, such as exist in some fevers, &c., receive the name of *leucocytosis*. If leukæmic blood be kept standing some time, the red corpuscles will sink, forming a dark lower layer, above which the white corpuscles float in the serum as a pus-like upper layer. The white cells in leukæmia appear to us to be of larger size, and to have bolder nuclei than ordinary healthy white cells. It is said that in leukæmia from disease of the lymphatic glands, the cells are smaller; but we have no experience of a purely lymphatic leukæmia. This changed state of the blood has a curious effect on the microscopic appearance of the organs. The capillary vessels are found stuffed with the white blood-cells, so that these often serve to mark out the course of the minute blood-vessels in a very useful and interesting manner. We have already described some of the effects of this state of the blood on the liver, where the white cells leave the vessels, and form collections along their course. The same occurrence takes place on a smaller scale in other organs, especially the kidneys, the wall of the intestine, and the heart. In these organs, pale, roundish patches are found, which are very conspicuous to the naked eye; but when the microscope is used the effect is disappointing; one only sees that some of the white corpuscles have escaped from the blood into the tissue. We thi better not to call these patches leukæmic tumour. The term leuk tumour belongs to tumours of which but few instances are reco none have as yet come under our inspection. In the cases related, t were considerable lymphoid formations in the alimentary canal kidney. These are figured by Virchow in his 'Geschwulste.' We seen such formations as are there described in cases of precisely sin structural character, which were shown by Dr Murchison, at the Pa logical Society, in 1869 and 1870; but in none of those w have so come under our notice was any leukæmia found. number of observations attesting the presence of leukæmia with t tumours is small; and notwithstanding the care and interest with w they have been sought in later years, few cases have come to li hence it might be questioned whether these malignant growths be to leukæmia; some examples have, however, been very convincing.

It is said that the blood in leukæmia contains a large quantit certain chemical principles, which were found by Scherer in the spl tissue, especially hypoxanthin.

It is important to notice that although leukæmic patients ultima may become very anæmic, yet the disease is often found in people do not look anæmic. We have seen several cases fatal before anæmi in; the veins often look full of dark blood, and the lip has a pur colour in leukæmic individuals, and this when they may be near de for although the mode of death is generally by a gradual was some cases have died unexpectedly and almost suddenly. T Recklinghausen gives an example in which death occurred twenty- hours after a full meal; and you will remember the death that occu here recently in the case of a leukæmic woman, who was sent into hospital for abdominal tumour. She was not thought to be very ill, certainly was not anæmic, yet she died after a few hours' illness, q unexpectedly. In neither of these cases was apoplexy found, altho this is said sometimes to cause the sudden death of leukæmic pers In our case the state of the heart appeared to be the immediate c of the unexpected death; the ventricles showed some pale patches f extravasation of the white cells of the blood.

Atrophy.—Besides finding the spleen immensely enlarged, we meet with it equally surprisingly shrunken; it may be reduced to exceedingly small size. It is said to have been found weighing only drachm, and many cases are recorded of its having only the size walnut, &c. Under these circumstances the usual varieties of sistence are met with. We do not know that any symptoms att on this atrophy of the spleen; it occurs especially in old pers Considerable wasting of the spleen is natural in age.

Softening.—We have several times alluded to the variations of consistence of the spleen associated with its variations of size, but it will be needful to direct your special attention to the extreme softening of this organ, which is apt to occur in febrile states. This softening is generally, but not invariably, accompanied by enlargement. The softening may be extreme, and may reach such a pitch, that the splenic pulp is almost diffluent, and the whole organ like a bag of thick purplish creamy liquid. In this condition it may rupture during life. The microscopic examination of such spleens is very difficult; nothing is discoverable that can give an explanation of the change; it is the pulp that is softened and not the trabeculæ, so that a stream of water easily washes the pulp out of the trabeculæ, and thus these spleens serve well to display the trabecular structure. We know no ground for the statement of Rokitansky and others, that the spleen in fevers contains a peculiar deposit, the so-called typhoid deposit. It is a point of interest in reference to the relation of the spleen to fevers, that the blood in typhoid is found to contain some excess of white cells. In a living case of typhus, the other day, we carefully compared the number of white cells, in the blood just drawn, with the number present in the blood of four healthy persons, and we found the typhus blood to contain at least three times as many white cells as the healthy blood. This case proved fatal.

Melanic Spleen.—*Melanæmia.*—As a result of intermittent fever, pigment is found in the spleen and other organs, especially in the liver, brain, and kidney, as well as also in the blood. There are reasons to think that the spleen is the source of the pigment, or at least the chief source. The reasons are chiefly these: First, the spleen is known frequently to contain, under ordinary circumstances, cells which hold blood corpuscles in their interior in a more or less defaced condition, and also to contain pigment grains and flecks in its pulp. Secondly, the spleen is commonly severely implicated in intermittent fevers, which cause melanæmia; nevertheless, we cannot conclude that the spleen is the sole source of the pigment, since in a case observed by Frerichs, the pigment was found in the liver, and not in the spleen.

There is little to describe in these spleens, except the change of colour, which becomes either greyish-black, or bluish-black, or dark brown. The consistence varies as usual in all splenic diseases, being softer in the early stages, and denser in the latter; but this rule is subject to the accidental cause of the patient's death. There is generally some enlargement of the organ.

Pigment is found both free and enclosed in cellular bodies of various forms, of which some resemble the spindle-shaped corpuscles that line the splenic vein, and others are of a more rounded figure. The free pigment also varies much in its physical condition. In well-marked examples of the disease, the blood is found to contain pigment in much the same forms. The discovery of the pigment in the blood, which first threw light on the nature of melanæmia, is due to H. Meckel. It is believed that the pigment formed by the spleen passes from this into the portal blood, in which it reaches the liver, where its coarser particles lodge in the outer zones of the lobules; the finer particles pass through the liver, and are found in various parts of the body, not universally. Thus, they do not discolour the muscles nor the skin; at least, not constantly or characteristically, though a sallow hue of the complexion is often present; but the brain and kidneys show the black colour very frequently. Thus, the process may be described as microscopic embolism of the affected organs, with pigment produced in the spleen. In many cases observed in America by Drs Meigs, Rhoads, and Pepper, the pigmentation was found to be limited to the portal blood.

In this country little attention has been given to the disease. It is, indeed, rarely met with here, since the conditions of severe and prolonged ague, to which melanæmia owes its origin, have been almost entirely dispelled by the drainage of marshy places in our island. We have met with but two examples of this disease, and can add nothing to the already very full descriptions of it; but great interest attaches to the drawing here shown you: it is a sketch of a brain from Dr Bright's 'Medical Reports,' which undoubtedly represents this affection. The case is here described as that of J. C—, an old man, who, accompanied by his wife, set out from Horncastle, in Lincolnshire (an aguish locality), in August, 1829, for London; and having walked all the way, fell ill immediately on his arrival, and was brought to the hospital in a dying state, apparently with fever. On post-mortem examination the brain was found as here represented, having its superficial cineritious structure of the colour of black-lead, but the section showed the medullary part unaffected, except the puncta vasculosa, which were found of the same blackish colour, and the vessels were filled with dark blood. The spleen was also of a dark hue. The wife's brain and other organs were also dark, though less so than those of her husband.*

Although the anatomical facts concerning melanæmia are well established, and their interest is very great, yet there are conflicting opinions as to the importance of the disease. Thus, Niemeyer argued against its significance, on account of the fact that the cerebral and other symptoms of severe intermittents, which one would account for by melanic disease of the brain and other organs, show variations in intensity, especially in the intermissions that characterize this class of diseases; also they are often cured by quinine. This inconstancy of the effect, he urged against a belief, that a constant condition like melanæmia could be the source of the trouble. But this argument is quite inconclusive, because it is in the nature of the intermittent fever itself to bring with it the alternations in question, and if any organ is diseased by the melanæmic complications, that particular organ will show symptoms of unusual disorder during the paroxysm, although in the absence of the paroxysm its melanic state alone is insufficient to create serious disturbance. In the absence of the paroxysm the brain does its duty indifferently well, although there are always present malaise and indisposition to exertion, &c., in the intervals; but the paroxysm brings out the weakness of the diseased organs, and may well be expected to reveal the relative severity of the melanic disorder by the relative intensity of the symptoms; thus explaining why it is that the pigmented change in the brain has been found deep in proportion to the violence of cerebral symptoms in intermittent fever.

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* The decease of Dr Bright since this was written leads us to notice the merits of this great physician, which should be studied and imitated by us all. We allude to his truthfulness, and his publication of all the facts relative to the cases which came before him; even without knowing their import or that they contained any discovery. This he did in his account of renal disease; subsequently, in describing pancreatic disease; and now, after thirty years, we can make use of one of his cases, of the pathology of which he was ignorant, but of which he gave all known particulars, even to the place whence the patient came, which is now the most important part in the history, and yet, probably, has been read by hundreds of persons without any conception that the mention of such a fact could bear on the nature of the case.

Enlargement of the Splenic Corpuscles.—We find great varieties in the size of these bodies in different cases, but it is doubtful whether they have any pathological significance. Experiments have shown that these corpuscles, or "Malpighian bodies," vary with the stage of digestion, and this will sufficiently account for the different sizes they present to the eye, sometimes being large and at others invisible. One point of interest respecting them, is their very great size in some fatal cases of purpura which occurred a few years ago.

Injury.—This is not uncommon in injury to the abdomen; if the splenic vein be opened the blood may drain from the vena portæ, when the first thing noticeable is the black pitchy character of blood effused. The spleen may be burst open from unequal pressure on its surface, or may be transfixed through the diaphragm by fractured ribs. Injuries of the spleen are by no means necessarily fatal, for cicatrices may sometimes be found on this organ; and in cases of accident from falls, &c., which are not immediately fatal, and in which the spleen has been lacerated, the various stages of repair may be seen. Here is one with lymph on the surface a week after the injury, and in this other case 5 oz. of laminated clot, in process of absorption, are found lying on the surface of the spleen. The death occurred eighteen days after the accident, and was caused by abscess of the brain.

We have already alluded to reported instances of spontaneous laceration of the stomach and liver from violent efforts during vomiting; but there can be no doubt that the spleen may give way from undue distension, such as occurs in intermittent fever; there are many authenticated cases of the occurrence. That which, however, is, perhaps, more frequent in India and other hot climates is, that persons suffering from this morbid enlargement receive a slight blow, and this is the immediate cause of the rupture.

Fibrinous Deposits or Embolic Infarction.—As we have already said, two different conditions of spleen prevail in heart-disease according as the disorder is obstructive or inflammatory. In obstructive heart-disease the spleen is dark and hard; it, indeed, partakes in the indurated state which is common to all the great viscera in cardiac ischæmia. In acute endocarditis with ulceration, on the other hand, the spleen is generally soft and large, and it is apt in these cases to show certain local diseases which are known by the term "embolic infarctions," or sometimes "fibrinous deposits." These embolic infarctions appear on the surface as pale patches abruptly circumscribed, and generally situated in the anterior thinner border of the spleen. There may be but one or two, or the number may be very great; their size too is subject to much variety; when cut through they are seen to dip into the tissue like wedges, this shape corresponding to the distribution of the vessels, for if you examine our specimens of the partially injected spleen, you will find that when the small branches only have been filled, the portions injected correspond in their wedge-like form with the patches we are speaking of. Similar deposits are found in the kidneys, in the same class of cases, and it is possible that the sub-pyæmic condition already alluded to may have some intimate connection with the change in these organs (see Embolism). The appearance of the deposits depends on the stage at which they are found; when first formed they are paler than the surrounding texture, and are surrounded by a narrow deeply congested zone of tissue which defines them strictly and plainly. If the artery of the affected part be opened, or its section examined, it will be found to be full of ante-mortem clot. In the course of a few weeks from the time of its first formation the patch grows gradually paler and paler, acquiring at the same time a characteristic yellowish or brownish tinge, and at last the portion shrinks so that its surface sinks below the general level, and the tissue around it falls in until finally a scar of fibrous tissue may be all that is left. Scars along the anterior edge of the spleen often have such an origin, as you will prove by observing how frequently they are associated with ancient heart-disease.

This is the course taken by the simpler examples of embolic infarction, but in some cases more severe results arise. Thus, instead of a tissue going through the above simple wasting, the patch shows signs of inflammatory softening or it suppurates, this change being accompanied by peritonitis of the capsule of the spleen. Such a result we have several times seen in cases of ulcerative endocarditis. When this is the case the same severity of the course of local change coexists in any other examples of embolism that may be present in the kidney, brain, or other organs.

We believe the cause of these infarctions is the entrance of clot into the arteries of the spleen, and that the variation of the action in the part, depends on the nature of the clot introduced. If it be clot formed passively in the heart, &c., by simple deposition of fibrin, then the infarction leads ultimately to wasting, but if the embolus is a fragment from an ulcerated spot, then it sets up inflammation, even to suppuration, in the seat it reaches. We must then distinguish two sorts of result—Firstly, those due to the obstruction of the artery; the consequences of this occurrence have been worked out experimentally very fully; shortly they are these: the distal part of the obstructed vessel and the vein corresponding, become distended, apparently by the vessels suffering malnutrition and losing their power of resisting the general vascular tension; you know this tension is shown by the hæmadynamometer to be very considerable, hence dilatation of the weak vessels and stagnation of the excess of blood in them; while the blood leaks through the vessels and fills the interstices of the tissue, and the tissue itself deprived of its natural supply and flooded with extravasated blood fails in its nutrition and undergoes involution and absorption. The vessels in the borders of the affected part partake somewhat in the distension that thus affects the part itself, yet the blood in them does not undergo coagulation and involution, hence it is seen as a dark zone of congestion around the patch. Secondly, the changes due to inflammation are those ordinarily seen in that process, but complicated by the effects of this circumstance, that the inflamed part has its vessels plugged, the result of which is that the inflammatory activity is generally limited to the zone around the plugged part, which is apt to separate in a more or less necrosed state, forming thus an ill-conditioned abscess, into which the living parts around, partaking in the inflammation, continue to discharge pus.

The milder form of this process is what we generally see in heart disease, the severer is identical with what we find in pyæmia; indeed, it may be taken as a description of the ordinary course of pyæmic abscess of parenchyma, which many of the best authorities believe always arises by embolic plugging of the part. When these embolic abscesses form in consequence of inflammation in the left side of the heart they may be well called by the name of *arterial pyœmia* to distinguish the whole case by this title from the more common cases of pyæmia, due to surgical sores and wounds, the difference being that in the latter cases the disease generally affects the theatre of circulation of the venous blood, while in arterial pyæmia the secondary suppurations are found in the theatre of distribution of the arterial blood. The mechanism of the local process is, however, the same in both cases. Embolic infarctions of the simple kind uncomplicated with evident inflammation are rarely met with except in heart disease. It is still a question how they are formed. We have expressed our belief that they are embolic, but some think that they can arise by coagulation of the blood *in situ* in the affected part of the organ or by some obscure special changes not clearly conceived. The reason for such belief is that infarctions are met with in some cases of morbus cordis, where no source of emboli can be found. It might be thought that they then correspond to apoplexy of the lung, supposing that the blood bursts out into the tissue in that occurrence.

Where no vegetations were present in the heart, it is possible that such vegetations might have been formed and swept off, but this is a hypothetical escape from a real difficulty, and against it is the further fact that in some other conditions in which the heart is not specially diseased, the same infarctions have been found. Thus, in fever, both typhoid and relapsing, Dr Murchison has shown cases of these infarctions, and the same has been seen by ourselves both in relapsing and typhoid fever. How shall we account for this? We believe it arises by the formation of ante-mortem clot in the recesses of the ventricle or auricle during temporary feebleness of the heart's action, these clots whilst yet recent being thrown into the tissues on the speedy recovery of greater power by the heart. In favour of this view we will get you to reflect how frequent it is for us to find ante-mortem polypi in the heart, in persons who have long had feeble circulation; these people may live on for months, as is shown by the advanced softening changes the clots undergo, but no doubt such clots form to a less extent under conditions of less extreme weakness than exists in these more obvious cases; slight clots very probably form in fever and in heart disease, for we cannot suppose that every ante-mortem clot produced in the ventricle keeps its place there, and we might thus anticipate that where recovery takes place while the clot is yet loose, the loose clot is easily cast off into the blood. There are no examples recorded which are not explained by this hypothesis; we believe that all cases of so-called thrombosis of the arteries arise in this way by embolism with recent clot, whether they be the cases found in puerperal women or cases called thrombosis in the cerebral arteries, such as are recorded by Dr Dickinson ('St George's Hospital Reports').

The microscopic appearance of these infarctions reveals the persistence of the splenic tissue in them, and the gradual disintegration of it by a fatty transformation, while a quantity of hæmatoidin crystals are always formed in it. This presence of blood-crystals, together with the superficial seat, and wedge-like shape of the patches, forms the chief means of distinguishing hæmorrhagic infarctions from other local diseases of the spleen such as tubercles, Hodgkin's deposits, &c.

Inflammation and Abscess.—In describing embolic infarction we have mentioned the fact that when the embolus comes from an inflamed endocardium into the spleen it sets up inflammation in the embolized spot; the result being that an abscess forms there, which has all the characters of position and outline that belong to these infarctions, especially in that they are found at the surface and usually at the thinner edge of the organ, dipping into the tissue in the form of a wedge.

Abscesses of the same general characters arise from common pyæmia, such as we meet with after surgical operations. These pyæmic abscesses have quite the same appearance as the embolic abscesses. Often all the stages of the embolic and suppurative processes can be seen in the same spleen. The earlier stages are exactly alike in the two conditions so as to suggest it as probable that pyæmic abscesses are also embolic, and take their origin in particles carried from the primary sore; this theory we have elsewhere considered. The abrupt demarcation of a part of the tissue by a deeply congested zone, while the included portion becomes gradually paler and more opaque, is common to both conditions, but soon the pyæmic part softens down into a cavity containing pus, in which are fragments of the original tissue in a state more or less broken down and detached. Some cases show more severe extremes and change than others, in some gangrene may occur; the gangrenous condition is generally dependent on a gangrenous state of the sore or wound that causes the pyæmia.

These abscesses are generally not large, though by their number, and by breaking into each other, they may occupy more than half the spleen. The peritoneum over them is coated with lymph, but it is very rare to see general peritonitis set up in this way. You know that a general pleurisy is often occasioned by pyæmic abscess of the lung. But the peritoneum differs from the pleura in this, that it covers many organs with various functions, and it is worth a moment's notice how much more often an intense inflammation near the pleura creates pleurisy, than similar conditions succeed in creating peritonitis. There are exceptions enough, it is true, but we never saw pyæmic abscess of the spleen create general peritonitis; twice, however, we have seen chronic abscess of the spleen set up suppuration limited to the lesser cavity of the peritoneum, the foramen of Winslow being closed.

It is a question whether idiopathic abscess of the spleen ever occurs;

we have met with a few cases of long-standing abscess in the organ, but it was doubtful whether they had not an hydatid origin; one of the cases was from a sailor who had recently come from the tropics. Yet the spleen is not unfrequently found involved in suppuration.

Among other cases which are very obscure during life are some which are found to be due to localized suppuration in the left hypochondriac region. Circumscribed abscess in this part we have now seen several times; in some cases it has been due to a chronic ulcer of the stomach. In two or three there has been a communication with the intestine, although whether this was the primary or secondary affection has not been very clear; we have seen also one or two instances of suppuration in this region touching the spleen where no such cause could be found. We mention these cases that you may not be unprepared when you meet with them. Lower degrees of inflammation of the spleen are very difficult to recognise; it is not unfrequent to find the peritoneal coat of the organ more or less inflamed, and coated with plastic lymph, and we still more frequently meet with the same process in a later stage, when the spleen is adherent to parts around, especially the diaphragm; generally no further peritonitis is present, and hence we are almost obliged to conclude that the inflammation belonged to the spleen itself rather than the peritoneum. In this way we may infer splenitis from perisplenitis, but the conclusion is not so satisfactory as if we had obtained direct evidence of inflammation of the tissue of the spleen, for we occasionally meet with perihepatitis, without inflammation of the liver. We have, however, on one or two occasions met with diffused subacute splenitis, the affected portion of the spleen was more solid and deep coloured than the rest of the organ; and the whole spleen was much swollen and adherent to its bed. This happened in the body of a person affected with syphilis, who had a similarly diffused subacute pneumonia. For further particulars see 'Pathological Society's Transactions,' 1871. On the whole the character and means of recognising the lower degrees of splenitis are very unsatisfactory, and stand much in need of further investigation. The condition of the capsule should be carefully observed; a little recent lymph spread over it easily escapes notice. We have seen such a layer of recently formed lymph on a soft spleen, in a case of death from rheumatic fever.

Chronic Capsulitis.—*Chronic thickening of the capsule.*—We have just mentioned the frequent adhesion of the spleen to the diaphragm; this is generally found quite unexpectedly, but it often occurs in the bodies of persons who have suffered from tropical fevers. We believe such adhesions are an important cause of pains in the left side; having met many instances in which pain in the splenic region was much complained of by persons who proved to have adherent spleen. The pain is no doubt due to the resistance such a spleen opposes to any cause of distension during portal congestion. Besides these adhesions, the capsule of the spleen rather frequently shows circumscribed thickenings of various sizes, and more or less diffused over the surface, but usually limited to the convex side. These often reach a surprising thickness, and then closely resemble common fibro-cartilage. Section shows them to be laminated parallel to the surface, and the microscope reveals a fibrous structure, the fibres being arranged in dense areolated lamellæ. Such semi-cartilaginous thickenings of the splenic capsule usually occur with similar thickenings of the capsule of the liver, and we consider them to be among the most decisive evidences of chronic alcoholismus that post-mortem examination reveals. A minor degree of the same thickening is occasionally found, marking the surface of the shrivelled spleen in an elderly person.

Apoplexy.—We may here mention this form of disease, which proves rapidly fatal in domestic cattle, and has the character of an acute fever, the spleen being found distended with extravasated blood and enlarged to an enormous weight. Bacteria are said to have been discovered in the blood. We have no knowledge of the occurrence of this disease in the human subject.

Lardaceous or Waxy Spleen.—We have already spoken of lardaceous liver, and the spleen may be affected in a similar manner; indeed, it is one of the principal seats of the affection. In most cases the amyloid matter is not uniformly diffused, but affects the Malpighian corpuscles, which become greyish and semi-pellucid, so that on section they give to the organ an appearance that has been well described under the name of the "sago spleen," the corpuscles looking like boiled sago-grains scattered through the tissue. On applying tincture of iodine thinly to the surface of such a spleen, these corpuscles become in a few seconds stained of a deep brown colour; this dark colour, as you know, is the characteristic reaction of the amyloid substance in lardaceous disease.

The red pulp of the spleen in these cases is so little affected that the microscope is required to reveal the amyloid change, but thin sections soaked in iodine-water show under the microscope the small arteries to be characteristically coloured. In the same sections the state of the Malpighian corpuscles can be more closely examined, and it will be seen that in them the amyloid change is not confined to the small blood-vessels, but has extended from these to the lymphoid structure which composes the parenchyma of the corpuscle, converting it into the usual glistening mass which readily absorbs iodine, and acquires a characteristic deep brown colour.

Such a microscopic examination by the aid of iodine is always required to discover the lesser degrees of lardaceous disease, for it must be remembered that the customary application of tincture of iodine is but a coarse test. When the Malpighian corpuscles are in the above-described sago-like state, the spleen is not generally large; but sometimes no sago-grains appear, while the lardaceous spleen is large and of a homogeneous appearance on section. As far as we have observed, Malpighian corpuscles are always absent in these uniform lardaceous spleens; when present the corpuscles are always first and chiefly implicated. The consistence of a lardaceous spleen is almost characteristic, its peculiar feel, resembling that of firm lard, will generally be recognised after it has once been carefully noticed. Like all lardaceous organs such a spleen is anæmic, and has a peculiar translucent and lustrous look on section, is capable of being cut into thin knife-like slices, and has an indescribable peculiarity of soft toughness, very closely resembling the consistence of raw bacon. It is necessary to remember that lardaceous matter never occurs free, but that the amyloid matter is most intimately associated with the structural elements, being within the component cells of the arteries, &c., and causing these cells to swell; hence the unfortunate impossibility of obtaining it pure for chemical examination.

Tubercle.—Tubercle is met with in the spleen in two forms, first as minute grey granulations, sometimes extremely small, not larger than grains of pepper, at others approaching the size of millet seeds, and then having a more yellowish colour. This form of tubercle is always found as part of a general tuberculosis, and in size, &c., the tubercles correspond to those found in the other parts of the body. When small and grey such tubercles are with difficulty distinguished from the Malpighian corpuscles, but we have observed that a stream of water will decide the question. The Malpighian corpuscles are always softer than the rest of the spleen, and so wash away first. Tubercles, on the other hand, cling firmly to the trabeculæ, while the pulp is washed away.

The other form of tubercle is found in large yellowish deposits, scattered about in the organ. It is much more rare than the miliary kind, and its pathology is less definite, cases occurring which present characters more resembling tumour-formation than tubercle, while others belong to syphilitic gummatous products. Precise criteria for the definition of these several conditions are indeed wanting, but in determining the nature of any particular case, we should be guided by these characters as distinctive of tubercle: first, a generally rounded figure of the masses, which vary in size; second, an almost complete caseous transformation of the deposit; and, third, the presence of miliary-sized granulations forming the recent growing outer edge of the patch, which, if large, may be softening down in the centre.

It is probable that these large tubercles in the spleen are primary, that is, belong to its proper pathology, while the miliary tubercle is always secondary to some other tuberculous source.

Hodgkin's Disease.—This name is useful as distinguishing a diseased condition common to the spleen, the lymphatic glands, and often to the liver, which was first pointed out by Dr Hodgkin. It constitutes a disease that can be recognised clinically, and is not unfrequent. The glands are found enlarged, and containing the same formation as that in the spleen. The state of the spleen is very remarkable. It is increased more or less in size, and contains a number of deposits of yellowish-white tolerably firm opaque matter, resembling at first glance masses of suet, scattered through the organ, or much more rarely collected into a larger mass in it. The masses are generally of angular outline, of very various sizes and embedded deeply in the spleen-tissue. They are connected with the trabeculæ of the spleen, and have once been observed to radiate from its vessels, a few reach the surface, and sometimes a granular formation of the same substance is found in the capsule. The deep seat and the absence of hæmatoidin pigment distinguishes them from embolic infarctions. The structural characters of the patches vary somewhat, but the microscopic appearance generally shows a fibrous meshwork containing more or fewer lymphoid cells, *i. e.*, the structure known as "lympho-sarcoma" or "adenoid" tissue, or more roughly as "fibro-nuclear tissue:" sometimes the fibres preponderate and the masses are harder, sometimes the cells are vastly more numerous, and then both in appearance and in tendency the diseased patches closely resemble cancer, so that they may spread to parts around malignantly. But some of the cases graduate towards tubercle, becoming caseous, and softening down, or being associated with general tubercle. Others have much more the character of lympho-sarcomatous tumour.

The association of these formations in the spleen with similar growths in the portal canals of the liver, and the glands, producing gradual œdema, and wasting, with dropsy, mark a clinically important disease. It may occur at any age; thus the last two cases we met with were aged fifty-four and sixty-one years respectively, but it is more common in young people.

Cancer.—Excluding cases of the kind we have described under the name of Hodgkin's disease, primary cancer of the spleen is exceedingly rare, and the spleen is also very rarely affected by secondary cancer. When cancer is widely diffused in the body we may find the spleen implicated, masses of growth rising from the surface or embedded in the interior. These secondary masses of cancer have a rounded figure. This spleen exhibits within it a melanotic tumour, such as existed elsewhere in the body from which it was taken. The cancer is usually a soft and rapidly growing sarcoma or carcinoma.

Hydatid, and other Cysts.—Hydatid of the spleen is not co case occurred here some years ago, where a very large attached to the spleen, as in this drawing; it was apparentl Old calcareous hydatid cysts have been met with in the splee specimen, which has been thought to show hydatid cysts, the or three cavities within the tissue of the organ, and thus it be rather a growth or cystic formation resembling that in which we have already shown you.

DISEASES OF THE SUPRA-RENAL BODIES

The great discovery which will deservedly perpetuate the name of Dr Addison has had the effect of directing the attention of the profession very strongly to the diseases of the supra-renal capsules. You are aware that his observations showed that when the supra-renal bodies are destroyed by disease a gradual loss of constitutional power ensues, accompanied in many cases by a remarkable discoloration of the skin, which has been called *melasma* or *bronze skin*. It was long a question of great interest whether the destruction of the capsules is the immediate cause of the symptoms of Addison's disease; and, although we are in possession of very definite facts bearing on this question, yet the conclusions indicated are not so clear and precise as could be wished. The principal facts and conclusions may be shortly stated thus: first, the fatal symptoms which constitute Addison's disease are almost always caused by a very chronic and complete destruction of the whole of both capsules; second, it is remarkable that no other disease of the capsules, such as cancer or lardaceous change, produces the symptoms of Addison's disease. Here is a dilemma, for if all destructive diseases of the capsules do not produce the disease, there must be something peculiar in that special disorder of the capsules which does produce it, or else there must be in that special disorder an implication of organs not affected in the other destructive diseases. Of this dilemma some prefer the one arm and some the other. Of those who affect the first some ascribe the peculiarity of the symptoms to the peculiar constitutional character of the morbid change, being impressed by the really interesting fact that *other tuberculous diseases produce melasma;* thus, phthisis of the lungs or kidneys sometimes induces considerable darkening of the skin; other reasoners would set down the peculiarity to the slow rate of the change as compared with cancer; others to the greater completeness of that change.

On the other part there are great authorities, who are more disposed to adopt the other arm of the dilemma, and to look altogether away from the capsules and the process in them, and consider the proper symptoms of Addison's disease as due to implication of the nervous connections of the supra-renal bodies, especially of the solar plexus; relying as a material basis for this view partly on the fact that the tuberculous inflammation causes fibrous thickening of the parts around the capsules, and partly on a single case observed by Bell Fletcher, where it was thought that symptoms of Addison's disease resulted from disease of the head of the pancreas. This is the view which Virchow favours; it has very recently been worked out by Risel in an interesting paper, in which he gives his reasons for believing that the abdominal sympathetic is affected by contiguity with the capsules, so that the vaso-motor nerves of the abdomen are paralysed; hence, he thinks, the blood accumulates in the abdominal viscera disordering their functions, while it is withdrawn from the rest of the system so as to cause anæmia of the brain and insufficiency of supply to the heart, and thus to induce the lassitude and feeble pulse of Addison's disease. In considering how far such a view as this includes the truth we must remember that the capsules themselves are highly nervous organs; their medullary part is full of sympathetic nerves with ganglia, and their connections with the solar plexus are numerous and large, so that in the capsules themselves we have nerves enough to make their destruction important from a point of view regarding sympathetic paralysis. But as to the positive observations of implication of the solar plexus, although fifteen have been collected, yet these vary so and are often so ill-defined, while microscopic observations have several times shown the nerves sound, that but little reliance can, we think, be placed on this source of evidence at present.

Meantime, while we believe that it is through an influence on the abdominal sympathetic that the fatal symptoms of Addison's disease are developed, our opinion is that the essential importance of the supra-renal capsules themselves in the disease has not been touched by any of the arguments adduced. For although cancer of the capsules rarely, or according to some never, produces Addison's disease, yet the force of this is taken away when we remember that likewise enormous cancer of the liver occurs without jaundice. Also as to lardaceous disease, if it be true that when it affects the capsules it does not produce melasma, so likewise is it true that when the liver is ever so lardaceous, neither jaundice nor ascites will result unless some other disease be present, yet no one, hence, is led to cast doubts on the connection of jaundice with liver disease in acute atrophy, &c. Hence we are justified in saying, that the relation of Addison's disease to the capsules is no more obscured by its comparison with cancer and lardaceous disease of those organs, than is the relation of acute atrophy to the liver obscured by its comparison with cancer and lardaceous disease of the liver. In either case there is a sufficient explanation of the persistence of function, in the fact that cancer does not entirely destroy the organ affected, and the parts that are spared are able to fulfil completely the duty of the whole organ, being, as far as the cancer goes, quite healthy. Indeed, it is matter for surprise how rare it is for cancer of any vital organ, to destroy life by suppressing the function of that organ, *except by mechanical obstruction or pressure*; for a small proportion of the substance of any of the parenchymatous viscera is adequate to discharge the whole function, as one may see in the case of those examples of chronic granular kidneys, in which persons pass for healthy, until an apoplectic fit carries them off, and their kidneys are found reduced perhaps to one fifth their proper weight—mere defaced relics. We must then remember, in order to hold firmly the correct conclusion, that in all cases of uncomplicated fatal Addison's disease, both capsules are totally destroyed by a very chronic and gradual process. We attach no importance to the experiments on rats, because in the first place the conditions of digestion, &c., are so vastly more complex in the more elaborate nervous organism of man, from whom even bad news will take away appetite; and in the second place the preserved rat did grow prematurely old, and bald, and miserable, whilst Addison's disease is itself very chronic and slow, outlasting in man the length of life which the rat enjoyed after the supposed removal of his capsules.

Inflammation.—When suppuration exists in the neighbourhood of the capsules, they may become involved in it; one such case we have described in which the capsules near a carious spine, were enlarged in all their dimensions, so that they were about twice their natural size; they had a deep purplish-red colour, without any of the usual outer opaque yellowish zone. No special symptoms were observed in this case, but several cases have been met with of hæmorrhagic inflammation of the supra-renal bodies, bilateral in Virchow's cases, unilateral in a case by Koehler, this state of the capsules being the chief morbid change in the body, when the patients had died with typhoid symptoms.

Apoplexy.—The supra-renal bodies are liable to suffer extensive extravasations of blood; this may happen either on one or on both sides. Two cases of bilateral apoplexy of the capsules are recorded in the 'Pathological Society's Transactions,' and in one other shown by Mr Jessop, of Leeds; there was tuberculous suppuration of one capsule, and a large apoplexy into the other. In these cases the effusion is into the medulla of the organ, so that the cortical part is distended into a cyst containing the blood. The medulla of the capsule is, as you know, very frail, and so easily breaks down, indeed, that it is often found to be crushed in removal, so as to form a large cavity surrounded by the cortex. This artificial cavity originally gave the name of "capsule" to the organ. The supra-renal body shares this extreme fragility with the substance of the brain, and, like the brain, it becomes liable to suffer an extensive laceration of its sub-

stance when its vessels, insufficiently supported, have at last giv
the blood extravasated meeting with little resistance.

Addison's Disease—*Tuberculous inflammation.*—The conditio
capsules in this disease is much alike in the several cases. The
are enlarged and hardened, and more or less firmly adherent t
around. Their surface is nodular or uneven, and the proper s
almost lost. There is often no discoverable trace of the
structure, but its place is taken by two kinds of material, of wh
one which makes up the greater portion, and is more centrally
is more or less like hard cheese, being opaque and yellowish-wh
toughish or friable. The other substance is in lesser quantity,
more in the outer part; it is grey and semi-translucent, h
slightly pinkish appearance when exposed to the air. In some
the cheese-like substance there are often spots softening dow
puriform fluid, while in other parts of their substance a qua
lime-salts are deposited, converting the organ to a stony or cre
mass in such part or parts.

It is evident from this description that so far we are deali
the remains of a bygone activity which has left indurated case
fibrous relics. The microscope quite confirms this impression
the caseous mass we find only granular detritus and defaced c
fibrils, and in the grey translucent part only some fibres with
lymphoid cells. Such caseous products are usually called by th
but significant name "scrofulous;" but to understand the pro
must find some portion of the diseased change in an active
otherwise we have no direct means of knowing its nature. S
direct light of great value as showing the tuberculous characte
complaint is, however, obtained in observing that Addison's di
nearly always associated with more or less of tuberculous di
the lungs. Thus, fifteen cases have been exhibited at the Path
Society; of these, twelve showed more or less tuberculous diseas
lung; in two the lungs were not examined, and only in one c
they said to have been found healthy. But besides this very
evidence of the tuberculous nature of Addison's disease, we h
direct observation of cases found in earlier stages, showi
presence of tubercles. We have before stated that in most
clinically uncomplicated Addison's disease the destruction of t
sules is entire; but there are some examples in which, when co
with pulmonary or renal phthisis, death occurs while yet the c
disease is in progress. In these circumstances the change is
found in the medullary portion of the organ, and it consist
production of grey tubercles which enlarge, fuse together, an
undergo involution, producing the mass of caseous and ot
degenerate tuberculous matter which we have before described.

The process is chronic, and lasts often several years. The die generally slowly by asthenia, but some have gone off suddenly by rapid sinking and syncope. The remarkable pigmentation of the skin gives the proper anatomical characters of ordinary dark skins, the pigment being in the cells of the *rete mucosum*, or, in darker examples, also in the superficial parts of the papillary layer of the corium.

No other organ exhibits any characteristic changes ; the blood is said to be sometimes leukæmic, anæmia is not always marked. Exophthalmic goître has twice been found. The heart is often atrophic ; but none of these conditions can be regarded as characteristic, or as throwing light on the obscure question, of the constantly mortal issue of the disease. The only reasonable suggestion is that which we have before mentioned, namely, that the disturbance of the abdominal sympathetic nerves in and about the capsule induces the fatal prostration. This disease is said to have been found in some of the lower animals, as the rat and the camel.

Tubercle.—Besides the tuberculous inflammation which constitutes Addison's disease, the capsules may be affected along with other organs with scattered tubercles in general tuberculosis ; this is, however, of little practical importance as regards the capsules themselves ; no symptoms are traceable to them in these cases.

Adenoma.—We have occasionally met with growths of supra-renal tissue, forming considerable tumours, say of the size of a chestnut, in or upon the supra-renal capsules. These larger tumours have always been single in each capsule, but once both capsules were affected. They have always been loaded with fat, but otherwise presented natural supra-renal structure. No ill results were traceable to their presence. It is far from unfrequent to find a greater or smaller number of little granular formations of supra-renal tissue studding the surface of the capsules, but quite separable from them. In this the capsules are like the spleen with its occasional spleniculi. This uncertainty in point of number in these organs refers interestingly to their embryotic development, which is by changes in the fœtal connective tissue, and not by any formation from pre-existing organs like the development of the liver and kidneys.

Cysts very occasionally occur in the supra-renal bodies. *Hydatids* have been met with ; once the hydatid was as large as an orange.

Tumour or Cancer.—Cancer of the supra-renal capsules is not very rare ; Averbeck has collected forty-three cases. When tumours are found in these bodies, their development is generally a part of a general malignant distribution of the tumours throughout the body. Under such circumstances any of the various forms of cancer may be met with. Cases of *melanosis* are recorded ; also of *malignant blood-cysts*. The majority of these tumours are called encephaloid, but when carefully examined they often prove to be *sarcomatous* in their proper histology; usually they are soft and of rapid growth. Occasionally cancer of the supra-renal bodies is primary; we met with a case not long since wherein both supra-renal capsules were greatly enlarged by primary cancer, each being as great as a fœtal head at full term; the structure was sarcomatous. Primary cancer of these organs is, however, very rare. The relation of cancer of the capsules to Addison's disease is discussed in our introductory remarks to this chapter.

Fatty Change.—The capsules vary exceedingly in the quantity of fat they contain in different subjects. You know that if you make a section of the recent organ you find the middle or central part composed of a pearly white substance, having a large vein in its midst; surrounding this is the cortical structure of a yellowish-brown colour, but composed apparently of two parts, since its inner margin, which touches the medulla, is of a much darker colour. It is this darker portion which is seen constituting the surface of the cavity when the organ has been torn open in removal. The dark pigmented interior then seen, gave rise anciently to the notion that these capsules were reservoirs of the black bile, or "*capsulæ atrabiliares.*" Histologically there is, however, no difference between the deeper brown layer and the more superficial buff-coloured layer, except that the latter is loaded with oil-globules contained within the cells that compose it. The amount of oil is enormous, comparable only to the quantity of fat found in very fatty livers. Equally with the fat liver there is no degeneration along with this fatness of the cortex. The oil drops are lodged within the cells without any apparent detriment to them.

The amount of this fat and the consequent thickness of the pale exterior of the cortical layer is very various; sometimes there is none of it, or only traces, forming little yellowish patches on the brown surface. This absence of fat has no constant relation to the leanness or obesity of the subject. The capsules are often fatty in lean people, and *vice versâ*. We have never been able to trace any physiological or pathological consequences of the varying amounts of fatness of these organs. The fatness cannot be called degeneration, for the tissue is integrally sound.

DISEASES OF THE URINARY ORGANS

KIDNEY

Malformation.—The commonest abnormal condition is the *absence of one kidney;* sometimes there is no trace of it, or its ureter. At other times some of the remains of the tissue are left; the latter fact suggests the same cause for its occurrence as for numerous other malformations already mentioned—that is, a morbid condition during fœtal life affecting the nutrition of the organ and causing its subsequent atrophy; for in these cases we find remains of the blood-vessels, and the ureter may be present, though extremely small. The suprarenal body does not share in the malformations or malpositions of the kidney. The kidney on the opposite side is enlarged; so that it is often equal to the natural size of both kidneys. Sometimes the two kidneys are united together over the spine, constituting the *horseshoe kidney.* The junction is always at the lower part, so that the semicircular organ has its convexity downwards, the blood-vessels enter on each side as usual, and the ureters pass down behind it. The union of structure may be complete, but in one case we met with a junction by merely fibrous tissue. The kidneys may also become altered in shape by a curvature of the spine, as you see in this specimen.

A very common abnormal condition of the kidneys is that in which they are *lobulated,* a state which is natural in the fœtus; the surface is seen to be deeply fissured and divided into distinct portions.

Here is an example in which both kidneys are united to form an unshapely mass, which lay within the pelvis. It has two ureters; but in a case related by Dr Delafield the two kidneys were united over the promontory of the sacrum, and there was a single ureter extending down and opening into the bladder in the median line. The vessels in these cases of low position of the kidney come off from the lower part of the aorta, or from the common iliac. Kidneys placed thus low may appear as tumours during life, and give rise to conjecture and apprehension.

But, besides the congenital malpositions which we have hitherto dealt with, the kidney sometimes moves from its place. This usually happens as a dislocation of the head of the right kidney from its natural position, wherein it is insinuated between the liver and the diaphragm, making, as you know, a shallow pit on the hinder margin of the liver. This displacement happens generally in women, as if through tight lacing, but it is often directly attributed to a strain. We have seen several examples, and in all these the kidney had turned so that its long axis was transverse and antero-posterior, the upper end projecting forwards between the colon and the liver. By pressure such a kidney may be made to cross in front of the spine, yet it is altogether behind the loose peritoneum, and has nothing like a mesentery. The displaced organ is said to have compressed the colon or vena cava, but there was no ill result in our cases. A movable state of the left kidney is much more rarely seen.

The position of the organs may be irregular in other ways; thus, occasionally, one kidney has been found situated low down on the psoas, or over the promontory of the sacrum, as you see here. We have seen two kidneys on the right side and none on the left.

Hypertrophy.—It is generally true that when enlargement of one portion of a viscus does take place, it is merely compensatory for the atrophy of another. Thus, if we find one kidney much larger than natural, we discover the other correspondingly small. These organs, however, may be often larger and heavier than usual, with evidence of congestion, in fever, pneumonia, &c. Or the enlargement may result from overuse; at least, we have several times found healthy kidneys to weigh as much as eighteen ounces in persons who have drunk enormously of beer; also in diabetes the kidneys are generally fourteen or fifteen ounces, that is, they exceed their proper weight by one half.

Atrophy.—The most common cause of general atrophy of the kidneys is chronic Bright's disease, an example of which we had last summer, where the kidneys together weighed less than an ounce and a half. Sometimes we find one kidney wasted, or scarcely a trace left, as we just now mentioned. We have met with some instances of small atrophic kidneys in young persons where the organs appeared to have been congenitally imperfect. Thus, in a young man of twenty-three years who died of chronic albuminuria there were but two pyramids in each kidney; and the kidneys themselves were very small, but without that granular and contracted appearance which usually characterises the small kidney in ordinary chronic Bright's disease; they were evidently deficient in development. We have seen degrees of this atrophic state so many times that we believe such a congenital deficiency to be an important predisposing cause of chronic insidious disease of the kidney in early life. You very often find local wasting of the kidney, its outline broken by gaps, in the depth of which fibrous tissue is found; some of these are evidently from old embolism, others from old inflammation and suppuration around the kidney, for not only may traces of such be found, but the subjects of the affection are commonly those who suffer from stricture, and are likely to have had nephritis; indeed, we have often had thus very sufficient proof of recovery from nephritis caused by stricture or stone in the bladder.

Injury.—Slight lacerations of the kidney may heal, especially in those cases where the peritoneum has not been injured, and any effused blood has been confined to the cellular tissue of the loin. Fractures of the kidney generally run in a direction vertical to the surface; in the course, that is, of the radiating pyramidal structure of the kidney. Hence there is often little bleeding, since the large vessels, having the same direction, are separated and not torn across. The urine, too, which is a most irritating fluid, scarcely escapes, and not in sufficient quantity to set up inflammation around; at least we are constantly examining the bodies of persons who have died several days after severe lesions of the kidney with extravasation of blood, without finding any products of inflammation. Sometimes the kidney is broken across, and its pelvis or ureter torn; in such a case there may be a simultaneous rupture of a vein, and thus much blood may pass down the ureter; perhaps coagulating within it, and forming moulds of its cavity, which escape in the urine, by which moulds this accident was on at least one occasion recognised by Mr Hilton during the patient's life.

Inflammation of the Kidneys.—You are already familiar with the wide difference between inflammations that are severe enough to destroy the structure of an organ, and those milder degrees that constitute catarrhs and inflammatory induration. In no organ is the distinction between these two branches of inflammation more important than in the kidney, or more evident. The destructive inflammations were known long ago, but it was left for Bright to discover and interpret the less obvious inflammations.

Suppurative nephritis.—This is the nephritis formerly so called when Bright's disease was unknown. It produces tangible results in the way of suppuration; and during life its effects on the urine, except so far as they are hidden by quantities of pus from the bladder, are too evident to escape notice. The urine always has in it unmistakable inflammatory products. But it is a secondary affection, that is, it never results from any of the forms of Bright's disease or other diseases which belong primarily to the kidney structure. It is always traceable either to inflammation of the bladder or to pyæmia, and hence it is often called the "*surgical kidney*," to distinguish it from Bright's disease, which comes under the care of the physician. Such a term is, however, not absolutely distinctive, for in the medical wards inflammation of the bladder often terminates in suppuration of the kidneys; especially the cystitis resulting from paralysis in spinal affections. In such cases as these, or in paralysis from fracture of the spine, or else in the widely different conditions of urethral stricture, calculus, or enlarged prostate, &c., the bladder becomes inflamed. This happens through retention of the urine until it acquires irritating properties, while the necessary catheterism adds to the irritation; the inflammation runs from the bladder up the ureter to the pelvis of the kidney; then an extension of the purulent process occurs until a suppuration arises in the cortical part of the organ. The mechanism of the extension is sometimes obscure; thus, you may find the ureters not evidently affected, or but slightly so, and they generally are not nearly as much diseased as the tissue of the kidneys. Hence some suppose that bacteria or some other means of conveyance carry up infection to the kidney.

If a patient has died with any of the diseases we have named you very usually find the kidneys much enlarged, and their surfaces more or less covered with minute points of pus, or small abscesses, and a section may show the suppuration to run down through the cortex of the organ, reaching to the cones, and often you can trace it on through these; but in cutting sections with this object you must bear in mind the waving course of the tubes, and then you will perceive that what appears on a section to be a deep-seated isolated suppurating patch is only the continuation of a superficial patch, down through towards the pelvis of the organ; a fortunate section will perhaps show you the suppurating tract in nearly all its length through the cones and cortex, and thus the distribution of the disease plainly traces the history of its spread from the pelvis of the kidney upwards. The abscesses rarely reach any size, so as to form a collection of pus beneath the capsule; and it is very rare for the peritoneum covering the kidney to be involved so as to set up a peritonitis. In all these cases the mucous membrane of the renal pelvis is inflamed in the same way as the urinary passages below, constituting *pyelo-nephritis*.

Suppuration of the kidney is a very grave addition to the danger of cystitis, but there is no special symptom by which it can with certainty be recognised in the presence of the already existing disorder of the urine; it has to be inferred from the gravity of the general condition.

The renal pelvis may, however, be inflamed without the substance of the organ being necessarily implicated. The mucous membrane is red, vascular, covered with a muco-purulent secretion, and not uncommonly by flakes of adherent lymph. For this inflammation the term *pyelitis* is used.

It happens sometimes that the surface is more than usually involved, and the capsules of the kidneys, with the adjacent parts, may be those especially inflamed; to such a condition the term *perinephritis* has been given. In acute cases of this kind we find the capsule highly vascular, while pus forms around it, producing abscesses which may seek an opening in the loins or into the colon, &c. But it is more especially as a chronic affection, or as a result of a former inflammation, that we would direct your attention to this, because it is often overlooked. You find, for example, on attempting to take out a kidney, that it is firmly fixed in the loin, so that the organ often slips out of its capsule, which is left *in sitû*, very much thickened, and having much indurated cellular tissue on its exterior, closely involving the adjacent fat; the surface of the kidney itself is rough or puckered, and perhaps torn by its separation.

Perinephritis, both acute and chronic, is sometimes caused by blows or other injuries to the loin, but it is mostly found in the same class of cases where the acute suppuration exists, viz. cases of stricture, &c. There can be little doubt that the adhesion indicates a former inflammation of the organ, and from the contraction of the substance of the kidney it is possible that even pus has sometimes been formed. Such a process probably accounts for the wasted kidneys sometimes met with; thus, in cases of stricture which we have examined, the kidneys have been much wasted, both organs presenting localised wasting, distributed so like the nephritic suppuration in stricture cases, that no doubt it arose in attacks of nephritis which had occurred several years before.

In cases of *general pyæmia*, especially those caused by periosteal abscess, small deposits of pus may be found in the kidney, as well as in other organs. We have noticed also that in cases of pyæmia from perineal section or lithotomy, &c., the kidneys show a greater proportion than usual of abscesses, although no sign of pyelo-nephritis be present. This fact is interesting, showing, as it does, that organs already having a partial cause of inflammation fall more readily into pyæmic suppuration. Such facts are not sufficiently borne in view by those who regard all pyæmic abscesses as embolic in their origin.

Abscess of the kidney is generally scrofulous, and we shall speak presently of this form of disease.

Bright's Disease.—It was known before Bright's observations that the urine is albuminous in some forms of dropsy, but he first demonstrated that this albuminuria is due to disease of the kidneys. Such kidney-diseases as produce albuminuria have ever since been called by the general name of Bright's disease; and although the renal changes which agree in producing albuminuria present, as we shall see, several distinct varieties, yet the characters which they possess in common unite them into a practically well-recognised kind. It is not so very surprising, as at first might appear, that these states of kidney should have long escaped notice; for the kidney itself gives usually no pain, and the urine is clear, while death occurs under widely various circumstances of dropsy, acute inflammation, apoplexy, &c., not one of which points evidently to the kidney as its source. The discovery and proof of the common cause of the vast number of different disorders produced by this disease, will ever make Bright's name illustrious.

We have said that the changes in the kidney which produce Bright's disease are various, but they are not so many as the crowd of names proposed for them; it is unfortunate that so very many terms are employed in speaking of this disease, terms which are incongruous and cannot be brought to correspond with each other. Some names regard the general appearance of the kidney; thus, you constantly hear of the chocolate kidney, the mottled kidney, the large white granular, or contracted kidney, the lardaceous kidney, &c. These terms are simple and explain themselves, but others are more ambitious and serve to embody theories of the nature of the disease often taken from disputed analogy or doubtful microscopical observations, so that they conflict more or less, and perplex the inquirer seriously. Thus, the names "*acute desquamative nephritis*," "*tubal nephritis*," "*acute diffuse nephritis*," "*croupous nephritis*," are all used for the same disease by distinguished authors, respectively by Drs George Johnson, Dickinson, Delafield, and Niemeyer.

The plan which the College of Physicians in its nomenclature has adopted, of calling all those conditions, so open to dispute, by the general name of acute and chronic Bright's disease, is that which we have always followed; it is clinically convenient, and involves no doubtful theories.

Acute Bright's disease.—Typical acute Bright's disease occurs as an independent ailment, associated with albuminuria and dropsy, often caused by cold, and then usually during exposure while the person is hungry or fatigued. It is often called *acute renal dropsy* or *acute albuminuria*.

But besides the independent disease that would thus be universally called acute Bright's disease you meet with a similar condition as a result of *scarlatina*. About the end of the second week of scarlatina the urine is apt to become albuminous; this may pass off without inducing serious effects, but sometimes, especially if exposure is allowed during convalescence, acute renal dropsy sets in, and when once established is apt to run the same course as, or perhaps a rather more favorable course than, acute renal dropsy from exposure to cold. Renal dropsy thus arising after scarlatina, easily allies itself etiologically with acute renal dropsy from exposure, if we believe that a chill to the desquamating skin is the cause of scarlatinous dropsy; taking such facts as those published by Dr Murchison in his reports of the London Fever Hospital, in which it appears that only a very small percentage of the scarlatina-cases had dropsy there, because the patients were prevented from leaving their beds until the third week; but we must not reason too readily upon such statistics and views, for it is known that in some epidemics of scarlatina the kidneys almost entirely escape, while in others they suffer very frequently.

But it is not only in scarlet fever that albuminuria occurs, it is very apt to appear in other severe febrile conditions, such as typhus, enteric fever, pneumonia, diphtheria, and many others. But it is remarkable that although albuminuria is frequent in these diseases, yet renal dropsy very rarely results from them. Also in chronic exhaustive diseases near their fatal issue, albumen may appear in the urine.

The mere *congestion* of the kidney, causing albuminuria, which accompanies many fevers, or is found in fatal chronic exhaustion, and which does not lead to dropsy, is altogether eliminated from acute Bright's disease by some German and American writers, these several states being grouped under the title "*chronic parenchymatous degeneration*." We think it is well to recognise strongly the distinction between this class of cases on the one hand, and those that prove obstinate and lead to dropsy on the other. But the name "*chronic*" and "*parenchymatous*" degeneration appears rather gratuitous for application to acute and temporary conditions probably caused by blood change. We have not ourselves found it a frequent thing for the kidneys in old cancer cases or other long-standing cases of exhaustion to present any more degeneration than belongs to the muscles and other parts which waste in the general wasting, so that we do not think this term "chronic parenchymatous degeneration" represents any improvement on the old way of recognising the albuminuria of fevers, &c., as due to to "*congestion*" or stagnation of blood in the kidneys. Such febrile *congestion*, however, has other factors besides the mere mechanical obstruction which is found in heart disease, &c., and then is also called congestion. For the blood in fevers is itself greatly altered, so that it stagnates in the skin as eruptions, or it even bursts out, causing ecchymoses; and in these hæmal changes the kidney, doubtless, has its share of disorder. But Dr Murchison clearly showed that albuminuria in typhus, fatal by convulsions, is due to a severe and evident congestion of the kidneys.

Under these circumstances of *febrile congestion* of the kidney the organ is found large, soft, and of a dark colour; on section the blood drips from it in greater quantity than natural. The microscope shows the epithelium cloudy, granular, or disintegrating in parts; and in places disturbed from its position, while in the channels of the tubes lymphoid cells are exuded, entangled, perhaps, in fibrinous casts; these are especially seen in the straight tubes. The interstitial tissues are quite natural, and free from inflammation, or swelling, or fat, or fibrous changes. Thus, the whole appearances are not those of a destructive disease of the kidney, but rather of a temporary disorder, corresponding to the quick recovery which follows if the fever is cured. Meantime the obstructed state of the tubes is a source of danger, and if it be extreme may lead to fatal uræmia and convulsions, as in Dr Murchison's cases. The state of the tubes is not equal; some you will find much more free and healthy than others.

It is of great importance to know what conditions of kidney are implied by such casts in the urine as are recognisable by the microscope during life, and whether such conditions are recoverable. In febrile congestion we find casts composed of lymph-corpuscles, blood, and renal cells, held together by an albuminous product, and such casts when found in more chronic cases signify an acute stage in the particular tubes they have come from; although in these chronic cases there will be also other casts from neighbouring tubes in different states, casts containing oily grains, fine granular casts, &c. In either acute or chronic conditions clear "hyaline" casts of fibrine may be found; these are often assumed to come from tubes that are denuded of their epithelium, but we have always found them lying in tubes with the epithelium perfect all around them.

Such *febrile congestion* is a long way from the *acute idiopathic renal dropsy*, yet there is a strong link between them in *scarlatinal albuminuria associated with dropsy*. Some regard scarlatinal disease of the kidney as a part of the general exanthematous disturbance proper to the fever, the kidney being affected like the skin and throat. Others look upon it as congestion of the kidneys caused by exposure of the recently peeled and tender skin to chills. The first view would approximate scarlatinous renal disease to other febrile disease of the kidney, while the second would make it identical with the acute renal disease from cold. It is probable, we think, that either view has a right application to particular cases; in other words, some scarlatinal albuminuria is like that of typhus, due to the febrile congestion, and this usually passes off with the fever, leaving no ill results; while other examples arise through chill, and thus come to partake of the obstinate character proper to albuminuria so arising. However it be explained, there is no doubt that while ordinary febrile albuminuria always disappears during recovery from the fever, and never causes dropsy, and while some scarlatinal albuminuria behaves just the same, some cases of albuminuria in scarlatina persist and cause dropsy. The renal dropsy of scarlatina is always recognised as acute Bright's disease. Indeed, it is sometimes very severe and even quickly fatal, so that the history of the earlier stages of acute Bright's disease is generally written from scarlatinal cases. For early death in acute renal dropsy from other causes is comparatively unfrequent, although, if the scarlatinal cases survive, their chances of ultimate recovery are better. Yet there is no difference in the appearance of the kidney itself, whether the cause be scarlatina or simple exposure to cold.

If you examine a case of about *three or four weeks'* standing you find the kidneys swollen and rounded, rather softer than natural, and oozing blood freely on section. So far it is like the febrile congestion we have described; but if you look closely you will detect a difference in that the texture is more opaque, especially in places. This opacity is hidden a good deal by the amount of blood present; hence it is better seen if the organ be soaked a few hours in water. However, there is less blood in the organs than in febrile congestion; you see on the section that the cortex is most affected; its Malpighian bodies appear as bright red dots. We think the great amount of blood present is chiefly due to the mode of death, by convulsions or coma. In one case of death by febrile exhaustion and asthenia in pyæmia with acute renal dropsy, the kidney showed little congestion, the change then was patchy opacity of the epithelium of the renal tubes, very well seen after soaking in water. This opaque white state of the tubes is, indeed, the essential part of the disease, and in doubtful cases it is a good practice to soak the organ before deciding as to its presence. The capsule is thick and opaque (but before setting this down to inflammation you must look at other fibrous membranes, and you will find that they all are thick and opaque through the dropsy); it strips off easily, and the surface of the kidney is then found smooth. The size of the organs and the mottling of them with white subopaque deposit are the essential morbid changes. The whiteness of the deposit is not like the whiteness of anæmia. It is due to fine grains in the renal epithelium reflecting white light; so it is of a creamy appearance, while anæmic whiteness gives the more pellucid appearance of the almost transparent renal epithelium. It is not evenly distributed, but some tubes are much more affected than their neighbours. But in this early stage, although the disease is essentially due to a whitening change, yet the amount of blood present, prevails so as to cause the colour of the organs to be at first sight of a deeper red than natural.

When, however, death occurs later in the disorder, as after *three or four months*, the kidneys are pale, even much paler than natural, while they still keep their large size. To understand why this is, remember, first, that the patient's blood has been much reduced by the disease; and, secondly, notice that the renal tubes have become so swollen by accumulation within them that they compress the capillaries between, and keep them nearly empty, so that the blood-redness is thus reduced. But meanwhile the fine grains of fat have increased in amount, and, by reflecting the light like any other white powder, have given more of the creamy opacity before spoken of. But the amount of fat-grains differs in different parts, and so some spots are very white, while around these the colour is greyish or pinkish and less opaque. The more opaque spots are usually small; they occupy the course of bundles of tubes, for they are the fat epithelium of the worst affected bundles, while neighbouring bundles are relatively free. Hence this stage or degree of acute Bright's disease offers you a *conglomerate* pale kidney. The white opaque clots were the "granules" of Bright, Rayer, and the earlier observers, and this form was their "granular kidney," but the name granular is now given to the tangibly granular, minutely nodulated kidney, formerly called "contracted" by Bright. These earlier authors thought the white granules a new matter effused in the parenchyma; but Bowman's discoveries were not then made, and the structure of the kidney was not known; the whiteness is not a new matter *between*, but a change of colour *in*, the texture, due, as we have said, to the powder-like white particles of fat crowding the renal epithelium.

Yet later, the progress of the same changes continues to make the organs whiter and even larger, until the classical "*large white kidney*" is fully developed. The surface is still smooth. This extreme of whiteness is due to the greater quantity of fat, and the greater and more general swelling of the tubes which expels the blood more than before. Thus, you see the whiteness is not due to passive anæmia, but rather it is *in spite of a tendency to congestion*, which it is important to notice and which reveals itself in three ways—1st, the pyramids are deeply congested; 2nd, you may find often effusions of blood into the renal tubes, forming red spots on and through the cortex, perhaps blackened by sulphur from the unsavoury neighbourhood of the colon, and then more conspicuous; they must not be mistaken for Malpighian bodies; and 3rd, you observe the veins of the surface engorged, their stellate and arborescent figures being often very beautifully conspicuous. The loading of these superficial veins will prove to you the pressure within the cortex of the kidney when you remember that these vessels are emptied by others that pass in through the cortex to the renal vein; it is the compression of these penetrating veins which dams up and so overfills these stellate veins, and they serve well to prove to you that the white cortex of the kidney has become white because it squeezes itself white.

These *congested*, *mottled*, *conglomerate*, and *large white* conditions are thus successive stages of acute Bright's disease. They are not at all distinct kinds of the disease, and by the accidental excess or deficiency of blood much variation of the appearance in any of the stages is induced. These stages used to be given as distinct "forms" of disease, and the effect was a tedious multiplication of kinds. Thus, Rayer gave six forms and Rokitansky eight. The numerous careful microscopic observations of late years have resulted in showing the identity of all these stages of acute Bright's disease.

The microscope shows a considerable degree of change, but yet it strikes you as remarkable that there is not more to account for the tenacious obstinacy of the disease. There is no destruction, as in phthisis; already with the naked eye you can see that the general structure remains unaltered. The disease, indeed, is founded in very minute disarrangements, so that the microscope may disappoint your expectations if they look for anything obvious. The chief changes are in the contents of the tubes. The epithelium is cloudy and opaque in the more translucent parts, and quite opaque in the more opaque white parts, of the kidney; you see tubes in these different conditions side by side. The regularity of the epithelium is disarranged, and some cells are detached, others disintegrated. The central channel is narrowed, or in some tubes obstructed, by being full of fibrin mixed with blood-cells, lymphoid cells, defaced epithelium, &c., but such obstruction does not affect all or most of the tubes. The tubes have been described as denuded of epithelium by desquamation, but we think this must not be too hastily allowed, for in making a section, however carefully, numbers of the tube-sections are emptied, and a single waving of a section through water will empty numbers more. If thickish slices be observed the deeper tubes appear to us to have their epithelium in them, and in very thin sections it is impossible to say that emptiness of the tubes is not through the process of preparation. The names "acute desquamative nephritis," "tubal nephritis," "croupous nephritis," &c., given to acute Bright's disease, rest on the belief that only the epithelium is affected. This belief is only an approximation to the truth, but it is true that the epithelium is *by far chiefly* affected. Nevertheless, we have always found in the conglomerate and large white kidneys that the intertubular tissue is coarse and fibrillar, and often loaded with fat-grains; to make the observation you must cut thin sections and wash out all the contents from the tubes by agitation in water. In rare and exceptional cases the Malpighian bodies are the parts chiefly affected; they appear as minute white points, and they are found stuffed, as in this specimen, with cellular formation. The reason of this is not known; usually the Malpighian bodies are only enlarged and engorged. The general analogies of acute Bright's disease in comparison with other diseases would perhaps be best expressed by the name *acute catarrhal nephritis*; its intense obstinacy is, no doubt, due to the impossibility of clearing the long tortuous tubes from catarrhal products. "Casts" are only found in some, not all the tubes; they are best seen in the straight tubes of the pyramids; they fill some of these tubes, which otherwise show comparatively little disease. These casts appear to us to arise by the semi-solid matter that obstructs the tubes filling them up, and projecting by a sort of overflow from the tubes, until having reached a certain length they break off.

Chronic Bright's disease.—Under this general name several distinct disorders may be brought. Firstly, the later stages of the acute Bright's disease which we have just described, namely, the large white kidney, would be called chronic Bright's disease, since the disorder that leads to it is often of many months' standing when it comes under fresh medical observation; but this inconveniently confuses acute and chronic; yet the College of Physicians call the large white the "fatty" kidney, and place it under chronic Bright's disease; as to this we shall presently show you that a kidney often is far more loaded with fat than in this large white state, without any Bright's disease at all. Secondly, the lardaceous kidney constitutes a chronic Bright's disease; we shall speak of this presently. But the typical and proper chronic Bright's disease is the *granular kidney*. This is chronic and insidious from the first, and is at no period acute, like the large white, nor is it a mere part of a general lardaceous disease in the abdomen, like the lardaceous kidney. We have already mentioned to you that Bright called our conglomerate kidney by the name of granular, and gave the name contracted to that kind of kidney we are now concerned with. In its typical form it occurs in circumstances totally different from those of acute Bright's disease. The patient dies from apoplexy, or acute pericarditis, or succumbs, as he otherwise should not, to some intercurrent accidental affection, there being no dropsy present, nor, indeed, anything to direct attention to the disease of the kidney if the urine should escape examination.

In this form of Bright's disease the tendency of the kidneys is to waste, so that they may be reduced in size until they weigh, as these do, only one ounce and a half. We generally find the kidney much smaller than natural, and with its capsule thickened and adherent. The surface not smooth, but on removal of the capsule seen to be irregular and minutely nodulated with small projections, which are now referred to in the title "granular" applied to the kidney. The general figure of the kidney is retained, but numerous irregularities of surface much deface its form. Its colour in a typical uncomplicated case, that has given no symptoms during life, is often not different from that of a healthy kidney, but it may be finely mingled with whitish, or greyish, or yellowish-white, through lodgment of fat in the tubules; when this is so the granular outline and strange colouring make the kidney look very morbid. Complication with acute Bright's disease we shall see has the same effect. Section shows the cortex to be thinner than natural; it may be extremely thin; it is not only thin, but its structure is seen to be confused or lost, so that the natural radiating striated appearance is not perceivable or is evidently destroyed; minute cysts may be seen in it more or less numerous. The masses of cortical tissue between the pyramids are less wasted; the pyramids themselves are not wasted at all. They are large—some think hypertrophied vicariously—and may show small chalk-like dots, which are minute calculi made of crystals of urate of soda—such urate as constitutes gout elsewhere; these crystals generally occur in gouty cases, and are thought to signify gout, even when there is no history of gout or sign of it in other parts; but this conclusion is not so secure as it would be if urates were not passing constantly down the tubes.

When the microscope is used then you see that the change is of a simple kind. The cortical tissue of the kidney is wasted, especially under the depressions of the surface, the elevations or granules being the best part of the organ, though the name "granular disease" of the kidney seems to level the accusation at them. The wasting is seen to be very unequal, so that some bundles of tubes suffer more than others. They suffer in two ways, some shrivelling up, almost or quite completely, while others are dilated. The shrivelled ones often in parts are reduced to scarcely traceable cords, but in other spots a small portion of their epithelium is preserved, and this epithelium appears as a round or oval body composed of cells enclosing a hyaline-looking mass. These bodies were described as cysts by Mr Simon, and were regarded by him as perhaps new structures, indicating a reconstructive effort to create new tubes; a similar view you will find has been held regarding the relics of hepatic cells in cirrhosed liver. We have often seen the wasted tube leading away from these little balls of epithelium, clearly showing their nature; the character of the cells and hyaline contents respond to those of renal epithelium enclosing a piece of cast. Other tubes dilate, and these are, perhaps, equally spoilt as to usefulness; the dilatation of tubes so tortuous forms cystic hollows, whose communications are hard to discover; these are the cysts described by Dr Johnson. The epithelium in them is so thin as easily to escape notice. They are sometimes comparatively large, and appear like minute spots to the naked eye, and by their number they then compensate for the wasting, so that the cortex may deceptively appear to the unaided sight not wasted at all. The loss of its structure must here guide you; it looks homogeneous, and has not its natural pyramidal striation. Some authors, especially Dr Dickinson, describe an increase of fibrous tissue; we think there is a little increase around the vessels, but not much. The patches, apparently of fibrous tissue, figured in Dr Dickinson's excellent drawings, we have always seen; but high powers resolve these patches into the remains of tubes, as, indeed, would probably follow from consideration of the drawings themselves. His drawing of healthy kidney has seven Malpighian corpuscles, but a drawing of granular kidney on the same scale, but of *half* that size, has twenty-one of these corpuscles, with but little tissue between; now, this shrinking to one sixth of the bulk is accompanied by dilatation of some tubes, so that a considerable space must be occupied by the compact walls of the necessarily numerous shrivelled ones. We have found that a fibrillated condition prevails in the tube-walls, which become coarse looking, but all large patches of apparent fibre we have always found to be chiefly made up of wasted tubes. The tissue of the organ is never tough, as in cirrhosis of the liver. The Malpighian corpuscles are sometimes said to persist and be enlarged, and we have found many of them certainly larger than natural, but the important change in these corpuscles is a wasting of them with the wasting of the tubes they supply. In bad cases we have found one third of them utterly wasted to little thick-walled corpuscles, recognisable by their artery. The blood-vessels are often excessively affected, not only the main renal artery, but its branches, which you may perceive projecting on the cut surface, and which you often meet with in microscopic sections, showing very thick walls. This hypertrophy of the arteries is partly, no doubt, due to the resistance which the shrivelled Malpighian glomerules offer to the passage of the blood. But a general thickening of the arteries of the body is usually present in these cases, and they generally show more or less evidence of degeneration of tissue as well as thickening. It is sometimes said that the arteries are apt to be lardaceous in granular kidney, but this is only as a complication, for when they are so the arteries of the spleen, liver, &c., are at the same time affected.

The epithelium varies much in its state; it may be almost natural in places; it is often full of fat-grains, or granular; in the dilated tubes it is thinned out much, being spread like a pavement epithelium on their walls.

The term *chronic desquamative nephritis* rests on a supposition as to the origin of the disease of which there is no sufficient proof. The title granular kidney involves no theory, and is in very general use. The pathological nature of the change is probably analogous to cirrhosis of the liver or sclerosis of the nervous centres. Some regard the epithelium as the special seat of disease; others the interstitial tissue, which is supposed to increase and compress the tubes, but no sufficient evidence of this is given. From having been often able to trace the shrivelled tubes up to wasted Malpighian corpuscles, we are inclined to believe that closure of these corpuscles may be the starting-point of the disease. But all that can be certainly said is that the tubes waste in the manner above described.

Such is the contracted granular kidney, the insidious disease of which we have spoken; but we meet with kidneys which have a decidedly granular or nodular surface and yet are large and whiter than natural. The granules are apt to be white, and the depressed parts between red. In a series of such kidneys some would vary in appearance towards that of the large white kidney, and others towards the granular kidney, so that you would call some of them large white examples of granular, and the others granular examples of large white kidney.

Now, what is the nature of these intermediate forms? Are they truly links which connect the large white with the granular kinds, so as to infer that all granular kidneys have passed through such stages from being large and white? This suggestion is negatived by the fact we have mentioned, viz. that the typical contracted granular kidney is not preceded by dropsy, &c., but is quite insidious. Experience shows that the granular kidney may reach its extreme without ever giving any alarming symptoms to the patient. Well, but suppose that in the course of this insidious process an attack of acute Bright's disease supervenes, such as would make a large white kidney. Then, undoubtedly, you would find a mixture of the large white and granular conditions. This is so very probably the explanation of the intermediate forms that we look for evidence to complete the proof; and this we think the microscope gives by showing that in them structural changes exist of exactly the kind proper to granular kidney, having just the degree and character which correspond to the extent of granular change on the surface; there is no blending of the two states microscopically, but an addition of the acute change. Thus, the granular condition in its first development implicates a few tubes only, especially those close to the surface; it becomes extreme in these, while others are free, and this limitation of place is very characteristic, so that it can be recognised with some certainty. Just such a limitation and distribution of the granular state do you find in the mixed cases, so as to show that the granular state has taken its usual course, and so to infer that it had its usual origin. The microscopic changes in granular kidney are much more marked than in catarrhal nephritis; hence these mixed forms are usually set down as granular by the microscopic observer, and we believe this view to be right so far that in these mixed cases the granular condition was primary, but we must not overlook the catarrh engrafted on it, as, indeed, the clinical history of these mixed states shows, for their clinical history is that of acute Bright's disease running its ordinary course, though we have observed that the course is shorter rather than longer when compared with that of the large white kidney.

Another question arises—Does the mixed form *ever* come through shrinking of the large white towards the granular form? May an approach to the state of granular kidney arise through prolongation of acute Bright's disease beyond the development of the large white kidney? The possibility of this cannot be denied, yet we have never met with any proofs of its occurrence. In all our numerous observations of these cases we have found the mixed forms of granular large white kidney to show the microscopic changes of granular kidney in their normal form, and not graduating towards the large white change, but mingled with it. The wasting of tubes and dilatation of other tubes with shrivelling of Malpighian bodies have shown just the same character and degree of development as corresponded with the granular appearance of the surface. There is no element introduced into the microscopic disorder that would induce one to suppose that the granular change arose in any other than the usual way. Thus, we do not find any evidence that the granular kidney arises from the large white. At the same time we do not deny the possibility of this. Thus, we regard the febrile congested, the mottled conglomerate, and large white conditions as successive stages in duration of acute Bright's disease. While the small granular kidney is a distinct disorder, running an insidious course, but liable to attacks of the acute form which produce intermediate conditions.

So far the matter is tolerably clear, but we have met with some cases that do not come within these descriptions, and as we cannot as yet explain them we will state shortly their circumstances. We have occasionally met with cases where there was no dropsy or history of dropsy, and where after acute suppression of urine, perhaps with symptoms like typhus, but without its rash, the kidneys were found enormously large, weighing 25 to 30 oz., and of a milk-white colour, perhaps the veins full of ante-mortem clot. The short course of symptoms, with the very large and white condition of the organs, are points requiring explanation. These cases are rare; the general appearance of the kidneys resembled lardaceous organs, but nothing of that change was present.

Lardaceous kidney.—Lardaceous disease is shared by other organs when present to any extent in the kidney; hence it is less properly a special kidney disease than the acute and chronic Bright's diseases we have described. Yet it leads to albuminuria, and, indeed, we have found indirect evidence that one at least of Bright's own figured examples of large white kidney was lardaceous, since the liver corresponding, which is preserved in the museum, we have found to be highly lardaceous. The lardaceous change was, however, unknown to Bright.

The kidneys are at first enlarged and pale; the cortex smooth, its structure not confused or disarranged, but well defined; the pale cortex contrasts with the dark pyramids. The consistence at this early stage is little altered. If tincture of iodine be applied to a section, in a few moments dark points and streaks appear in the cortex or at the apices of the pyramids especially, these dark parts contrasting strongly with the yellow iodine stain around. The microscope shows that these dark flecks are Malpighian bodies, or minute arteries whose inner coats are seen to be swollen and glistening, and to have become of a deep walnut colour by the action of the iodine. If no iodine be used the arteries look tortuous and glistening, the peculiar refractile lustre of the lardaceous matter prevents the microscope defining the minuter parts affected. The renal tubes are widened and their epithelium is flatter and thinner than natural. Often in the channels of the tubes are hyaline casts, which we have never seen to take the colour of amyloid matter by iodine, though other observers describe it so. The epithelium is perfect around these casts. In this stage the urine is very free and pale; at first without, or with only occasional albumen; gradually, afterwards, with much albumen. When the disease is long established and advanced the organ is, perhaps, shrunken again to its natural size, or smaller, and its outline is uneven, the capsule thick, in short, its state in this way approaches that of granular kidney. So, too, the microscope shows wasting, &c., of tubes, as in granular kidney. Also, now, degrees of catarrh may be noticed, and so the condition of large white kidney may be approached. Hence it is that some observers regard the lardaceous condition as only an accident supervening on Bright's disease. But against this view you will see that the peculiar characters of the early stage are decisive. For a long time the lardaceous state is independent, then, afterwards, upon it come the catarrhs &c., just as upon granular kidney catarrh may supervene. So it is that we get mixed forms. These arise, indeed, by attacks of catarrh upon kidneys in which lardaceous change is already advanced; and the change in question never progresses far in the kidneys without developing also in other organs. When this is the case the arteries and capillaries are much diseased, and the walls of the tubules in places affected; even the epithelium may be attacked, but this is more rare than is commonly described. The amount of lardaceous material now affects the consistence of the organ, which feels waxy, and looks dry and waxy on section, especially at the tops of the pyramids.

Lardaceous disease generally arises from syphilitic or strumous cachexia, especially when associated with suppuration. Any prolonged suppuration, as from old carbuncle or cancer, may cause it. Dr Dickinson hence calls it "depurative" disease, deriving the name from his theory of its origin, which assumes that lardaceous matter is probably dealkalised fibrin, and that pus, which is alkaline, carries off alkali from the blood, so that prolonged suppuration at last leaves some fibrin dealkalised in the vessels. Such views may constitute a step towards clearing up the great obscurity surrounding the lardaceous change; but it is not shown that by suppuration there is an absolute loss of alkali, and we meet some examples of very severe lardaceous disease where there has been no suppuration. This has been especially the case in syphilis.

Hydronephrosis.—In this condition the kidney is wasted and stretched about its pelvis, which is dilated through obstruction of the ureter. Thus a great cyst is formed, which may, from its size, be mistaken for a large ovarian tumour filling the abdomen, and an operation may be attempted for its removal under this belief. When in an extreme degree, the obstruction is commonly due to calculus impacted in the ureter, or to cicatrix of old disease in it due to passage of a calculus. But lower degrees are often bilateral, being due to obstructions in the urethra or bladder, or pressure on the ureters by tumours, &c., from without. The stages of the distension are interesting. Naturally the pyramids fill the calyces, but when obstruction causes accumulation in and tension along the ureter, pelves, and calyces, the latter widen and the pyramids shrink in a curious way, the circumferential drag of the distended calyx attached around the root of each draws it down so that it becomes flattened out at its base, while the apex still for a long while appears as a little prominence; at last this disappears, and in place of the pyramid a hollow is found, which hollow is continuous with the interior of the calyx. This occurs in each calyx until the kidney is spread out to form the walls of large cavities, the pelvis meantime dilating enormously, so that the wide calyces appear as sacs jutting from one side of it, the septa between the calyces being composed of the "columns of Bertini," which originally separated the pyramids from each other. Such hydronephrosis may suppurate through calculous or other irritation, and thus a large abscess or *pyonephrosis* form, causing adhesion of the contiguous viscera and tending to discharge itself in the lumbar region, or rarely by the psoas or colon, &c.

Cysts.—Cysts arising in the proper substance of the kidney are very different to the hydronephrosis thus described. These are of three different kinds:—First, there are simple cysts of no great importance frequently enough met with, especially in the bodies of elderly people; there may be one or more, but only a few, of considerable size, projecting in relief from the surface and sometimes large, the rest of the kidney being healthy and quite adequate to its office. They arise in the cortex of the organ. Their contents are mostly fluid, and when analysed are found to contain a small quantity of albumen, and the ordinary salts existing in the fluids of the body, but very rarely any urinary ingredients. Very often these cysts contain a thick *jelly-like matter*, sometimes like thin glue, and at others nearly solid, which resembles somewhat the colloid matter found in other parts of the body.

A much rarer kind of renal cysts, but of greater clinical importance, because the disease proves fatal, is where the kidney is converted into a mass of cysts, as you will see in several of these specimens, some of them holding several ounces of fluid; in these cases the renal structures may be reduced to mere relics found between the cysts; indeed, the septa between these are found to be composed of renal tissue when you would not expect it by their appearance. This renal tissue is then in the state of granular disease; sometimes a remainder of the substance of the kidney in its proper form is preserved on one side of the organ. Some of these cystic masses of disease are, as you see, ten times the normal size of a kidney, forming, indeed, a very wonderful pathological change. This pair of cystic kidneys weighed 8¾ pounds. We need not tell you that the name hydatid, which you see written on this old preparation, is erroneous; it was called hydatid because the term was used synonymously with cystic some few years ago. We shall presently speak of true hydatids in the kidney. These excessive forms of cystic disease may reveal themselves as tumours during life.

The clinical history is the same as that of chronic Bright's disease, of which, notwithstanding their vastly different appearance, these enormous looking tumours form only a variety. The cysts are, in short, an excessive production of that minuter cystic condition of the kidney which we have already described as occurring in granular kidneys. But there is another direction in which their pathology bears, which must be known. Of late years attention has been drawn, especially by Virchow, to a cystic change of the kidney, which is found in stillborn children, or as a congenital condition fatal very early. The kidney is found converted into a mass of cysts; this state often coexists with other malformations, as cleft palate, &c. It is a natural question whether the great cystic kidney of adults represents a lower degree of the fœtal cystic kidney which has proved viable. In favour of this view is the discovery of such cystic changes sometimes in young subjects, although the majority of them occur about early middle life. We have already suggested reasons for believing that some examples of small granular kidney represent originally ill-formed organs.

The last kind of cysts are those found in immense numbers and of microscopic smallness, which we have already fully described when speaking of granular kidney, to which they properly belong, and, indeed, of which they form the most characteristic feature.

Fatty Kidney.—When, a few years ago, the microscope was put into use to discover the nature of Bright's disease of the kidney, and fat was found in the tissue, an opinion was ventured that the disease was a fatty degeneration of the organ. To test the correctness of this, some diseased kidneys were submitted to chemical analysis, and found to contain no fat, when the opposite statement was made, that Bright's disease is not a fatty change. Both statements are true, but their application is, however, limited; the large white kidney of acute Bright's disease often contains a large quantity of fat, while the granular kidney of chronic Bright's disease may contain little or none. In the first case, however, the presence of fat is altogether a secondary affair, and due to a change or degeneration of the inflammatory products, and therefore the term fatty, as applied to the essential nature of the disease, must be as erroneous as it would be to call a cancerous tumour or a phthisical lung fatty because both contain an abundance of oil. We think, therefore, the term fatty kidney should be only used in the same way as we use the term fatty liver, *i. e.*, not for all morbid conditions where fat is found, but for those instances where the fat constitutes an independent diseased condition. Such a state is often met with in cases where the other organs are fatty; a small quantity of fat is generally present in ordinary kidneys, but in those we are speaking of the epithelium is loaded with it so as to be quite opaque in microscopic sections, and by reflected light to look creamy and opaque, but not very white, because there is no inflammatory accumulation, and hence no expansion of the tubes compressing the vessels. The function of the kidney in these cases may be disturbed, but, as in the similar example of fatty liver, the surprise is that with so striking a change in microscopic appearance so little vital disorder is evinced. Certainly there is no albuminuria or Bright's disease in these cases. As to the causes of this fatness of the kidney it occurs often in the large kidney of diabetes; in this it seems to correspond with the like fatness of hypertrophied hearts, which appears to represent the failure of nourishment of the overgrown elements before dissolution. It is also found in the general fatness or "steatosis" of phosphorus poisoning, and to a less degree in chronic antimonial and arsenical poisoning. It also occurs in some cases of great obesity, and sometimes we could not account for it. It must be clearly distinguished from Bright's disease.

Fibrinous Deposits or Embolic Infarctions.—These are of the same character, and have the same origin, as those in the spleen. We said, in that association, that such deposits are found in cases of heart disease, especially in those where the mitral valve is affected and its curtains covered with vegetations, whence it has been thought that some fragment from the latter, being taken into the circulation, is not arrested until it arrives at the minute capillaries of a parenchymatous organ; that there it lodges, obstructing the blood-vessels and producing a change known as capillary phlebitis. The method of production, however, is not yet universally agreed; all observers allow that embolism will produce these infarctions, and by all it is held as certain that an obstruction occurs in these parts, and that the vessels are blocked up, so that, owing to the impediment in the circulation, a stagnation of blood takes place in the affected part. In consequence of this the blood and the tissue undergo degeneration together, so that the red colour is lost from the centre outwards, while, perhaps, additional changes arise from inflammation. Thus, in the kidney, as in the spleen, a pale mass is seen surrounded by a dark red halo; the mass is wedge-shaped, owing to the mode of distribution of the blood-vessels, which correspond to the wedge-shaped radial segments of the kidney;

the base of the wedge is outwards, and, at first, slightly protruding, the colour at first dull purple, afterwards whitening, at first in the middle. If examined by the microscope the structure of the affected part is still seen to persist, but there is a granular or fibrillated material between the tubules, and these gradually lose their definition of outline. After a time the part begins to decay, it is more granular, and the tubes themselves are confused with granules. Here are specimens of the affection, and in this early volume of our 'Reports' it is figured by Bright and Barlow. After a time the mass shrinks until it quite disappears, leaving a cicatrix; and thus, in the same kidney, you may find instances of the affection in various stages—some wasting, leaving a depression on the surface; others not having altogether disappeared, and others which have left a deep cicatrix; you then see that the tissue amongst which they form becomes at last destroyed. Sometimes, though not commonly, they soften into a purulent fluid, but we have never seen a cyst produced, as some have described.

A plugging of the whole renal artery has occasionally occurred, with very different results in different cases. Cohnheim gives a case where he considered the effect to be a necrosis of the organ. We met with one where Bright's disease of both kidneys was already present, and there was a most interesting reduction of the disease in the organ whose vessel was obstructed. An example recorded by Mr Pick showed granular disease of the kidney as the result of such obstruction. In our case the cause of the arterial obstruction was embolism with an *ante-mortem* clot from the left ventricle.

Tuberculous Disease.—This is of two kinds: one, where miliary tubercle is scattered in the kidneys as well as in other parts of the body by general tuberculosis, is unimportant clinically, but offers a good opportunity of studying the origin of miliary tubercles. You see crowds of lymphoid corpuscles arising in the interstitial tissue of the affected spots; these spots are always small, and caseous change very quickly appears at the centre of the tubercle. The other form, which might be rather called a *scrofulous inflammation*, constitutes a primary and fatal disease. It is sometimes called strumous pyelitis, for the pelvis is soon affected. The leading symptom is a large amount of pus in the urine. After the continuance of the disease for some years the organ becomes quite destroyed; large hollow spaces exist in it with only a trace of the secreting tissue between them; the mucous membrane is thickened, covered with scrofulous matter, and beneath the membrane there is the same matter running into the tissue. The first stage of the disease is met with now and then accidentally; you find a caseous patch perhaps softened; it is situate just under, or at the surface of, one of the cones, or several cones may be affected. In other cases you find the attack to prefer the pelvis of the kidney, so that this is scattered over with tuberculous ulcers, the cones being little affected or even not at all.

These two modes of origin lead to two distinct developments in the more advanced stage. For in those cases where the pelvic mucous membrane is most affected there is generally soon some obstruction of the entrance of the ureter by inflammatory swelling or by an ulcer there. This causes a dilatation of the pelvis, and calyces, &c., like the dilatation which produces the "hydronephrosis" we have recently described. The remains of kidney tissue are then compressed, the pyramids flattened down, and calyces expanded, so that an enlarged and sacculated cyst, full of pus and lined with a tubercular suppurating tissue, is all that remains. We once met with such a cyst having a quantity of remarkably large and bright flakes of cholesterine in its contents. This is very rare; there are but few instances of it on record. In the other form the renal tissue suffers most severely, and although the pyramids, we believe, are always earliest and most severely affected, yet you may find in the thickness of the cortical part of the organ large vomicæ or caseous abscesses, which run together and increase and multiply till the whole organ is destroyed. You see by these specimens what a great destruction of the organ occurs. Sometimes you meet accidentally with examples where such a disease has been cured; of course the organ is destroyed, but the ureter has become closed, and the purulent and scrofulous matter, having been encysted, is now converted into a putty-like mass; when you meet with such masses accidentally in inspecting a body, you may regard them generally as dried-up abscesses. But sometimes the abscesses burst externally in the loin, or open into the colon. The disease is often unilateral, but after a long time the other kidney is apt to be affected. Sometimes you see miliary tubercles in the renal tissue around the scrofulous ulcers and abscesses in the pyramids. It appears that this disease is a primitive tuberculosis of the kidney, and so represents the primary renal phthisis; miliary tubercle, on the other hand, is always secondary. We have seen the primary scrofulous kidney set up general tuberculosis, just as pulmonary phthisis too often does. Its extension down the ureter to the bladder we shall presently consider.

Carcinoma.—As in general tuberculosis, so in general carcinomatous disease, you may find deposits scattered through the kidneys as well as other organs; but it is only after death that these are discovered. In this way sarcoma or melanosis or colloid of the kidney may be met with, as well as carcinoma. Where the kidney is the ordinary seat of cancer, there is usually a tumour, and the disease is recognised during life by the symptoms it produces. Yet in these cases it is remarkable how seldom the proper tissue of the kidney can be said to be primarily affected, for although a large tumour may exist, and this after death be removed as renal cancer, yet a careful dissection discovers the disease to have arisen primarily in the lymphatic glands, or other parts outside the kidney, especially about its hilus, while the organ itself is comparatively untouched; the disease, however, puts on the form of the kidney, for it has probably penetrated the hilus and expanded the capsule; this, we think, is the commonest plan of attack in primary renal cancer; it may be long before the pelvis of the organ is penetrated, and as the proper tissue is nearly intact, you can understand the frequent absence of diseased products in the urine. Occasionally, however, the whole structure may be infiltrated and immensely enlarged, as you see here, the kidney being converted into a large white mass. In the same way as biliary calculi and cancer are associated, so you may have renal calculi and cancer together, as is shown in this drawing.

Carcinoma is the kind of cancer most commonly present in the kidney. It is generally soft and *medullary*, as usual in vascular glands. Sometimes it rapidly enlarges through bleeding into its texture. You more rarely meet with the *scirrhous* form of carcinoma.

Lymphadenoma we have met with now several times in the kidney, associated usually with similar disease of the glands, and sometimes of the intestine and liver.

Leukæmic tumours we have twice seen in leukæmia, as small scattered roundish patches, which, however, resembled patches of extravasated white cells as much as actively growing tumours; they have, however, been seen in a less equivocal form as round actively growing tumours.

Fibroma is often found in the form of small white knots of fibrous tissue near the bases of the pyramids. These are generally quite unimportant; but twice we have seen them as large as a horsebean. In these cases some spindle-cells were present in the tumours. Once we have met with a very large simply fibrous tumour of the kidney.

Cavernous angioma is much more rare in the kidney than in the liver, but is occasionally met with there.

Syphiloma, or syphilitic gumma, we have once met with. Here is a drawing of the specimen; but gumma in the kidney is curiously rare. Very few cases are on record.

Villous disease of the pelvis of the kidney, similar to that in the bladder, has been very rarely met with; in a case by Dr Murchison there was villous disease both of the kidney and bladder.

Parasites.—*Hydatid* disease of the kidney is not very common. We often have patients before us with renal tumours in whom it is suspected, and you will occasionally hear of cases where hydatids pass in the urine; but in the post-mortem room they are not often met with; in two or three instances we have found them accidentally where no symptoms were noticed during life. They were on the surface of the organ, and had not interfered at all with its structure; in one of them the kidney was expanded around the cyst, but apparently quite healthy. In one case there was a hydatid in the liver, in the others the disease existed alone. In all of them echinococci were found.

Cysticerci have occasionally been found in the kidneys.

The kidney is the usual seat of ***Eustrongylus gigas***, the largest of the parasitic nematode worms; it rarely, however, occurs in the human subject; when present it leads to abscess and degeneration of the kidney affected. But it is frequent in many of the lower animals, especially weazels.

We would here also mention to you the escape in the urine of certain minute entozoa from the blood. Worms were long ago noticed in "chylous" urine; but it has been shown recently by Dr T. R. Lewis that these worms, which are named *Filaria hominis sanguinis*, are present in enormous numbers in the blood in these cases. It has yet to be discovered how they find their entrance into the body.

Calculi.—It is not unusual to meet with calculi in the kidney after death. In some cases both organs are found filled with them, the structure has been destroyed, and they have become moulded to the form of the pelvis and dilated infundibula; thus you see these large knotted and branching calculi fitting into the hollows of the kidney. They may be formed of uric acid, phosphate, and carbonate of lime, oxalate of lime, &c., and sometimes very beautiful semitranslucent white calculi are composed of cystic oxide. Occasionally they cause great changes in the kidney itself in the form of grave pyelitis and pyelonephritis, with the usual characters of these states before mentioned; or by inducing ulcerative or other inflammatory contraction of the ureter in passing down it; or else, by lodging in and obstructing it, they lead to dilatation of the pelvis of the kidney, which may be combined with inflammation, and in this way an enormous cyst containing pus may arise, which has been known to rupture into the abdominal cavity; or, this being prevented by adhesions to viscera around, the abscess has been evacuated by operation, or in any of the natural ways before mentioned in speaking of pyelonephritis. At other times you find that a kidney whose ureter is totally obstructed by a calculus has dwindled away to a small relic; whether this happens without any previous dilatation we cannot say.

URETER

Malformation.—One of the most common deviations from the natural condition is the presence of two ureters to one kidney, and this sometimes is associated with a double pelvis to the kidney; these two ureters may pass separately to the bladder, or unite before they enter. We have several times seen these variations.

Dilatation.—This is a condition very frequently met with, owing to an impediment to the flow of water to the bladder. Thus in stone, and various chronic diseases of the urinary passages, it is found; also from direct pressure on the ureters, as from ovarian disease, cancer of the uterus, &c. The dilatation is generally not uniform, but unequal, producing a sacculated appearance. The ureters are sometimes distended to the size of the intestine; numerous examples of this you will see on the shelves. When inflammation complicates the condition the ureter is thickened as well as dilated.

Obstruction.—This is often associated with dilatation, of which it is the cause; thus a twisting of the ureter, or stricture from previous ulceration, or else the presence of a calculus, will all produce these conditions. Here are specimens showing calculi in various parts of the canal. Some cases appear to indicate that a sharp blow on the abdomen struck over the ureter may cause inflammation enough to lead to subsequent obstruction.

Inflammation.—We have already said that in cases of suppuration of the kidney the disease has extended upwards from the bladder, so that not only the latter organ but the ureter is found to have its mucous membrane covered with adherent flakes of lymph, and pouring out a muco-purulent secretion. In some cases the coats are infiltrated, and consequently thickened. The signs of inflammation are generally less in the ureter than in the bladder or kidney.

Tuberculous Disease.—This is generally associated with similar disease of the kidney and bladder, as you will see by examining the preparations of these organs. The internal surface is covered with a scrofulous matter, the mucous membrane having long been destroyed, the walls are often extremely thickened, so that on section they look like a solid stem with a small canal running through its middle. This narrow channel can be easily closed by any fresh local swelling in its wall; and such an occurrence you will find indicated frequently in the clinical course of many of these cases, by the occasional augmented pain with reduction of the pus in the urine, such periods of greater pain being terminated by relief, coexistent with the reappearance of the free flow of pus.

Cancer.—In the same way cancer is not a primary disease of the ureter, but only occurs in connection with cancer of the bladder when the adventitious growth sometimes proceeds upwards in the course of this tube.

Injury.—The ureter generally escapes except in cases of ex
violence in which other fatal damage makes its condition

but sometimes gunshot wounds will specially select the ureter, as in the case of an archbishop of Paris. Very rarely, also, in a crushing accident, the force has by chance fallen destructively on the ureter, causing extravasation of urine in the lumbar region; we have seen two such instances. We have mentioned the occasional obstruction of the canal as a consequence of milder degrees of such injury. On the subject of injuries to the ureter you may read with advantage an able paper by Mr Poland in the 'Guy's Reports.'

BLADDER

Malformation.—The most common is extroversion of the bladder, accompanied by a malformation of the urethra; a fissure exists in the abdominal walls, the bladder is exposed, and the ureters are seen opening directly upon it, as you observe in these various specimens. The malformation generally extends up to and includes the umbilicus, and below it reaches and includes the genitalia, which are rudimentary and malformed, the pubic arch being incomplete. But in some cases the malformation more especially affects the bladder, the umbilicus above being pretty well formed, and the pubes and genitalia complete, so that the bladder forms a more or less protruding red pouch. The exposure of the bladder in these cases gives you an opportunity of examining the urine direct from the kidney, so that if you place a piece of test-paper to the opening of the ureter, you will find that the urine is strongly acid as it is poured into the bladder.

Hypertrophy.—This is a very common result of impediment in the urinary passages, and arises from stricture, enlarged prostate, &c. The muscular wall becomes much thickened, and on the inner surface of the organ the muscular bands are seen as strong cords interlacing with each other, not unlike the columnæ carneæ at the apex of the hypertrophied right ventricle. Whenever you find the bladder thus thick and reticulated within you should examine the urethra for stricture; low degrees of the condition are common enough; we often see a distinct but slight hypertrophy caused through the obstruction of the urethra in dropsy of the prepuce. If the hypertrophy of the bladder is efficient there may be no *dilatation;* this, however, very often accompanies hypertrophy, so that the organ becomes much enlarged in size as well as thick.

But in hypertrophied bladders it is far more common to find the organ *sacculated* than uniformly dilated. Sacculation of the bladder is a consequence of the distribution of its muscular tissue; this is not uniform, but the fibres are disposed so as to make a meshwork of strong bundles whose intervals are filled in with irregular weaker bundles. Under the greater pressure of the hypertrophied bladder when resisted by stricture, &c., the weaker meshes yield and the mucous membrane is forced out, at first but little, so that stretching of the bladder will efface the sacs; but afterwards they protrude outside the muscular layer, through its meshes, and reach and raise the subperitoneal tissue; thus numerous pouches may be formed chiefly in the sides and upper part, as you see here. The sacs are composed of mucous membrane covered by peritoneum, the submucous and subperitoneal tissues blended between them. Sometimes there is one large saccular dilatation, equalling the bladder in size; we met with one curious case where the bladder was severely inflamed, while a sac from its side, of greater size than the bladder itself, was quite free from the inflammatory action; upon these larger sacs you may sometimes see a few muscular bands running in their walls. Cases are recorded where great distension of bladder has been found in the fœtus, from some obstruction in the passage below. Sacculated bladders have been found congenital. These sacculi may contain calculi, which then are said to be "*encysted.*"

Simple or acute dilatation of the bladder is of great clinical importance. It is found in cases of fever or of injury to, or disease of, the spinal cord or brain, &c., conditions where the organ is paralysed and the urine retained. If this dilatation is not attended to it may lead to disastrous consequences in sloughing or, perhaps, rupture of the viscus.

Atrophy.—This is especially seen in long-standing cases of paralysis of the sphincter of the bladder, where the organ is never distended, but empties itself as soon as it receives any water from the ureter. Thus in cases of recovery from spinal paraplegia, where yet a constant dribbling of the urine is present, the bladder has permanently contracted, and become, as it were, merely a part of the channel between the kidney and urethra.

Injury.—The bladder is frequently cut in the operation of *lithotomy;* when we have met with such cases years afterwards we have been unable to discover the slightest trace of the wound. It is also frequently *punctured through the rectum,* to evacuate its contents, in cases of stricture, as you have several times seen. Besides more recent cases, three examples of some duration have come before us on the post-mortem table: in one, where death occurred a few months after the operation, the opening still remained between the two organs; in the second, where death occurred a year afterwards, the fistulous opening was with some difficulty found, it had not allowed any urine through it, except for a short time after its production; in the third case a small depression or hole existed just behind the prostate, but it was blind, and the wound in the rectum was perfectly healed, so that not a trace of it could be found.

The bladder may also be wounded by the point of the catheter. This is a very serious form of well-intentioned injury. In cases of cystitis after retention and catheterism you should always look well at that part of the back of the bladder which is opposite the urethra, and upon which the point of a catheter would impinge when introduced too far; you will then often see the cause of the cystitis. We commend this observation to your best attention. Injury to the bladder through accident occurs generally in cases of *fractured pelvis*, either from a heavy weight falling on the body, or a cart passing over it; the bodies of the pubes and ischia break, and the sharp ends may pierce the bladder, as you see here; or the bladder may be pierced by weapons, producing fatal extravasation, though a few cases recover, with urinary fistula.

Sometimes, as we have more than once seen, the bladder has been ruptured by similar causes, without any fracture, when blood and urine are poured into the abdomen, setting up fatal peritonitis; the bladder, when full, has also been burst by *direct violence*, as we have seen, from a kick in the abdomen; and there is reason to believe it has happened in the female during coitus. The most interesting question in reference to rupture of the bladder is, whether this can occur spontaneously from excessive distension; such cases are related, and this specimen is supposed to represent a rupture from such a cause; but it shows also, what we have always met with ourselves, a previously diseased condition of the organ. In the few such cases which have come under our own notice the history has been one of long-standing stricture, and subsequently of disease of the bladder; and after death a general or local peritonitis has been found connected with a sloughing condition of the latter organ, allowing the urine to escape through the coats. In these cases there was always a history of previous disease, which was sufficiently evident by post-mortem appearances; we have never yet seen a case of spontaneous rupture of a healthy bladder.

Inflammation or Cystitis.—We have already had some occasion to remark against the opinion which was formerly held too exclusively, that acute inflammations are the primary and typical diseases to which the body is liable; for, in fact, most of the changes in the body are chronic, and from these the acute diseases result secondarily. The term cystitis suggests these remarks, for, common as it is in connection with stricture, diseased prostate, calculus, and other chronic affections, it is very rare, if indeed it ever occurs, as an idiopathic acute disease. A calculus may set up cystitis and then itself escape *per urethram*. This has been held to explain some cases of apparently idiopathic inflammation associated with dilatation of an ureter. It is remarkable, considering the frequency with which the bladder is allowed to become overdistended, that severe inflammation from this cause is so comparatively unfrequent.

But cystitis caused by retention of urine or by foreign bodies will be found frequently enough; thus in persons who die with calculus in the bladder, or stricture, various degrees of *chronic inflammation* are constantly met with; the mucous membrane is sometimes destroyed in parts by ulceration, but more generally it is altered in character, thickened and congested, so as to be like red or purple velvet, or in severe cases it is covered all over or in patches with granular lymph, when the state receives the name of diphtheritic or pellicular inflammation. But you must not mistake for this the concrete layer of adherent mucus often mixed with phosphatic secretion, which is commonly found in the inflamed bladder. The graver changes are found at first on the summits of the rugæ, and always most advanced there. This appears to arise partly because the rugæ are kept exposed to the urine in all states of the bladder, whilst the recesses between them are closed by the coaptation of their sides; partly, also, it is due to the congestion which the summits of the rugæ suffer in comparison with the sides, which are kept more empty of blood by their coaptation and mutual pressure. The urine is purulent and alkaline. The most severe and *acute inflammation* of the bladder is generally met with in those cases where the organ has become paralysed from injury or disease of the spine, and especially if catheterism have been much adopted. In these cases the bladder becomes distended, the urine ammoniacal, the surface of the bladder inflamed all over, or, as we have already hinted, especially where the point of the catheter has touched it; it may have a slough at this spot. You may see here a case where death occurred a week after the first symptoms, and where the whole mucous membrane is detached, and hangs loose in the bladder. Cases are recorded where more or less of, or even all, the mucous membrane has been passed through the urethra. It is a question much discussed whether this intense inflammation and tendency to sloughing of the bladder (as well as the bedsore, which so often occurs in the same class of cases) is due to the nervous influence being removed from the part; whether it thus acquires its proneness to inflame, as some believe the cornea does when the fifth nerve is affected; or whether the inflammation be rather due secondarily to the paralysis, from this causing a retention and subsequent decomposition of the urine, which hence acquires irritant properties and tends to inflame the bladder.

Pericystitis is a term which we use for inflammatory processes around the bladder, in the subperitoneal tissue, and is not uncommon as a consequence of many pelvic diseases. The most acute form is seen from infiltration of urine, beginning at the neck of the bladder, and then pervading all the structures, so that when, at the inspection, the peritoneal coat is cut through, the cellular tissue beneath is found full of purulent matter, or even sloughing. Such suppuration may spread to the sheath of the rectus and other parts of the abdominal wall, and so require external opening. Thus sometimes from lithotomy such infiltration has occurred, producing this grave condition, and lighting up at last a general peritonitis. The same thing has been known to occur from pressure of a calculus ulcerating its way through the bladder. In cases of stricture, too, where the urethra is much diseased, so as to be perforated by ulceration or by the catheter, a like escape of urine may occur, with all the grave consequences we have enumerated, although then the perforation is in the urethra; we have examined a great many cases of fatal stricture and extravasation of urine, and, besides the subcutaneous inflammation, we have very often met with a similar inflammation, suppuration, or sloughing in the cellular tissue of the pelvis, behind the deep triangular ligament. This has been due to the mischief about the stricture proceeding backwards to the neck of the bladder, and involving the prostate in the inflammatory process, whence the disease extended around the bladder.

Instances have occurred where an abscess, arising from the presence of a calculus or other cause, has caused a fistulous opening into the rectum, or in the abdominal walls above the pubes.

In this specimen you see extravasation of blood beneath the mucous membrane, by which it is raised up into a number of projections resembling the surface of a placenta.

Tuberculous Disease.—This occasionally occurs as a primary disease in scrofulous subjects, but far more frequently it exists in connection with chronic tuberculous disease of the kidney. The latter organ is generally found in a state of more advanced disorganisation, having been first attacked by the disease; the ureter was next involved in the process, which thus spread down to the bladder. This is found thickened, its inner surface showing tuberculous ulcers, as you see in this preparation. The ulcers arise by the softening of miliary tubercles in the mucous membrane, just as in the bowel; hence they are at first small and round, but other tubercles form and spread and open in the floor and margins of the ulcer, so that it increases and becomes complex and deep with a thickened base. By the spread of this process the mucous membrane may be almost entirely removed, while the muscular coat is infiltrated with the tubercles.

Morbid Growths.—The bladder may be involved in cancer of neighbouring parts; this is especially frequent in cancer of the uterus. The back of the bladder may be laid open by this into the vagina.

The spread of cancer from the uterus is generally continuous through the coats of the organ, but we have seen nodules of cancer appear in the cystic mucous membrane when there was no continuity of this with the uterine cancer. We have seen the bladder invaded by cancer of the vesiculæ seminales.

Carcinoma in any of its forms may occur in the bladder. *Epithelioma* is rare: here is an example of it; it shows its usual characters in spreading locally and affecting the neighbouring glands, but not reaching remote parts. The mode of spreading of cancer in the wall of the bladder varies; sometimes it extends as an equable thickening, as in this specimen; at other times it forms a local mass that may project in a polypoid form. The surface of a cancer within the bladder, like that of cancers in the mucous membranes generally, may grow into villosities. Thus, in this specimen, where a thick mass is growing in the mucous membrane of the bladder, the surface is shaggy; and, on placing it under the microscope, it presents the appearance you see in this drawing, exactly resembling the villi of the chorion. Yet, as we shall presently mention, all villous tumours are not of a cancerous nature. Virchow has described a tumour with a stroma of smooth muscular fibre and nests of cancer-cells in the meshes under the name of *myo-carcinoma*.

Villous growth.—This is a very remarkable kind of disease; it occurs chiefly in young persons; small tufts grow on the mucous membrane, and cause death by hæmorrhage. We have now seen several of these cases; among others a railway porter, young and strong, was seized with hæmaturia, and in the course of a few weeks, in spite of all treatment, died from loss of blood. All that was found in the body were two small tufts scarcely larger than peas, and from these the fatal hæmorrhage had occurred. They appeared like little tufts of moss growing from the mucous membrane; and when examined by the microscope, presented villous processes, and what is very striking, as you may see in this drawing, which we made at the time, the surface is covered by columnar epithelium, long battledore-shaped nucleated cells, very different from the ordinary epithelium of the bladder. Each villus, we should have said, contained loops of blood-vessels. Another example was from the body of a woman; the whole patch was not larger than a shilling; the hæmorrhage was very profuse.

Polypoid tumours have occurred in the bladder; some have proved to be vascular, consisting of a deep-coloured mass of blood-vessels with but little to connect them. Others were of a fibrous texture (*fibroma polyposum*), others have more a *fibro-sarcomatous* texture. Small *fibrous* tumours form occasionally in the submucous tissue, especially near the neck; some of these partake of the muscular structure of the prostate gland and so are *myo-fibromata*. These polypi give rise to a fatal disease of the bladder, not, however, generally by hæmorrhage, as in the preceding case, but by interfering with the flow of urine, causing retention, cystitis, and consequent suppuration of the kidney. You have had an opportunity of seeing two of these cases lately, and they both occurred in children. So also in the specimens in our museum, which you see here, and in other cases of which we have read, the subjects have been children. You will see by these how the polypi grow within the bladder, and in considerable number, having peduncles, and being of a pyriform shape; their texture is soft, and composed of fibro-sarcoma; some of the smaller ones, you will see, have a warty character on the surface.

Foreign Bodies.—Our museum, besides showing calculi, has various other substances which have been removed from the bladder, both male and female:—pieces of catheter, slate-pencil, bodkin-cases, knitting-needles, &c., and these, you will see, are covered with phosphates.

Prolapsus of the bladder sometimes occurs in women during labour.

Hydatids.—Here are specimens of hydatids passed in the urine, but they probably came from the kidney; and this specimen shows a hydatid growing outside the bladder.

Bilharzia hæmatobia, a minute flukeworm, is found in the blood-vessels of the bladder, as a cause of hæmaturia, in Egypt and at the Cape of Good Hope.

URETHRA

Although the urethra is organized physiologically more as a genital than a urinary apparatus, and its diseases are caused generally in subserving its genital functions, yet pathologically the effects of these diseases tell more upon the urinary system, since, even if they interfere with the genital process, they do not so produce those serious consequences which they entail when they impede micturition; they then often prove fatal. It is for this reason that we place the morbid anatomy of the urethra with that of the bladder and kidneys.

Malformations of the urethra concern more the generative function.

Injury.—This generally arises from a fall on the perineum, which directly crushes the canal, or from fracture of the pelvis, in which the urethra is, perhaps, lacerated by a piece of splintered bone. But the frequency, nay almost constancy, of rupture of the urethra in cases of fracture of the pelvis, shows that it cannot arise in this way. And an explanation of its frequency is found in the close mutual connection between the bones and the urethra by means of the triangular ligament, so that by the disruptive drag upon this ligament, in which the urethra forms, indeed, the weakest spot, the urethra is torn when the pubic arch is broken.

Inflammation.—You may sometimes meet with a simple *catarrhal* inflammation of the urethra in boys, also in *gouty* persons. But urethral inflammation is mostly due to *gonorrhœa;* in this disease the inflammation commences at first in the anterior half of the channel and gradually passes more deeply until it may reach the region about the bulb, where it lingers long. It is said the endoscope shows that the persistence of inflammation in this part is due to a granular state of the mucous membrane, like that observed in granular ophthalmia or granular metritis. The endoscope affords great advantages over morbid anatomy in detecting the characters of these inflammations, for such characters nearly disappear after death, when the vital turgescence has subsided. Desormeaux describes an herpetic kind of urethritis known by its patchy distribution.

In the few cases we have seen post mortem the mucous membrane was deep purple in colour from congestion; the diseased appearance extended throughout the passage nearly to the bladder, and was strongly marked at the perineal bend of the channel; pus-like secretion was present on the surface. In mild cases, only the mucous membrane is affected, and that merely with catarrhal inflammation, but the inflammation may become very intense in the membrane itself, so as to cause a pellicular exudation on its surface, or a formation of pus in its substance, causing it to have a whitish appearance. Or the inflammation may extend to the fibrous tissue beneath or to the *corpus spongiosum*, or even the *corpus cavernosum*. In two grave cases we have seen phlebitis of the prostatic plexus, and pyæmia set up by this deep extension of gonorrhœa. Or the inflammation may spread to the bladder, or pass through the prostate and spermatic duct to the testes, causing inflammation in all these parts. This latter extension, however, belongs generally to the later declining stages of the disease, when the inflammation has reached the membranous urethra and is becoming chronic and granular.

A *plastic exudation* of lymph is not uncommonly met with in cases where there has been any grave injury to the canal, either from accident or in connection with operations. We more frequently found this in the post-mortem room in cases of old stricture, when there has been a necessity for laying open the urethra from the perinæum; here, perhaps from the incision, or more probably from the inflammatory process in connection with an extravasation of urine which has given rise to the necessity of the operation, we find the whole of the urethra, especially that part near the seat of the old disease, covered with flakes of lymph closely adherent to the mucous membrane, and sometimes forming a cast of the tube, or, instead of flakes of lymph on the surface, there may be pus in the substance of the mucous membrane.

Stricture.—Considerable obstruction to the passage of urine may arise through the changes in the urethra in acute urethritis, but stricture is essentially a chronic disease said to follow after a long course of granular urethritis; it is caused by a slow indurative inflammation of the submucous tissue, which hardens in becoming fibrous, and so constricts the urethral canal. You sometimes find this external induration extend into the cellular tissue all around, producing a hard tissue as dense as cartilage, and this is especially the case if connected with perineal fistulæ; sometimes the part closed is very short, so as to resemble a fold drawn across the channel, sometimes prominences project into it, sometimes a portion of the tube is simply shut up. About perineal fistulæ granulations may project into the urethra. It was formerly said, that the usual seat of stricture was the membranous part of the urethra; but it was pointed out, we believe by Mr Syme, many years ago, that the beginning of the spongy portion is its most ordinary situation. This is the most dependent part of the channel, and to this part inflammatory products would gravitate. Endoscopic examination proves that long after a gonorrhœa a granular inflammation lingers here. This part, also, is that which is most exposed to vertical pressure by the weight of the body in accidental falls upon the perinæum From examination of many cases of stricture we can say that it occurs either at the first part of the spongy portion or at the point of junction of the membranous and spongy parts. You never find strictures in the prostate, but they are sometimes seated towards the glans.

We will refer you to our shelves for the several specimens of stricture; in these you will see false passages passing about the sides of the urethra in various directions. In some cases there are many; we have found six distinct ways into the bladder in one example. Sometimes these false passages are lined by so smooth a mucous membrane that it is hard to say which is the proper urethra out of so many. Here is a specimen where the urethra is perfectly closed, the canal being quite obliterated.

Vascular Growths.—These occur mostly in the female, and are situated at the orifice of the urethra, forming a fringe around it. They are highly vascular and sensitive tufts, the villi containing loops of blood-vessels.

Carunculæ are spoken of as occasionally occurring in the male urethra, as small vascular growths on the mucous membrane, causing obstruction to the flow of urine. We have never yet met with this in the recent subject, although we have seen one or two so designated in museums.

Cancer.—The urethra may be involved secondarily in cancerous growths contiguous to it; thus, we have seen a medullary carcinoma of the bladder include the commencement of the urethra in its growth, and on more than one occasion the spongy part of the urethra invaded by superficial epithelioma.

Tubercle.—We have said that occasionally, in tuberculous disease of the kidney, the bladder is ultimately involved, and in one or two cases the commencement of the urethra has been also affected. In one of these the tuberculous ulceration of the membranous part had given rise to extravasation of urine. In this other example the tuberculous ulcers extend through two thirds of the length of the canal towards the orifice.

DISEASES OF THE MALE SEXUAL ORGANS

TESTIS

Malformation.—The testis may be absent on one or both sides, or its duct may be undeveloped when the testis is present. In other cases the testes are imperfectly formed and small. It is, perhaps, more important to notice that this organ on one or both sides may fail to descend into the scrotum, so that it is found in some part of the line it naturally travels from the loin downwards, unless it deviates and goes a little astray. Such retention of the testicle is more frequent on the left side. Its cause is not always discoverable, but sometimes there are peritoneal adhesions which explain it; otherwise we may suppose that the *gubernaculum* is not developed properly, or that the testis is too large, or the inguinal canal too small. When retained in the canal it may give rise to anxiety, and it is said, especially by Goddard, that through exposure in this situation and consequent liability to injuries, such testes offer a greater proportion of morbid growths than those which attain to their proper situation in the scrotum. He asserts also that no sperm is secreted by these testes. This was so in a case we examined.

Atrophy.—This may occur from actual disease of the organ, as will be presently mentioned, or simply from pressure on the cord affecting its nutrition; thus, after the long use of a truss, the testis sometimes wastes. The same may happen from a varicocele, or yet more frequently through the pressure of the fluid in a hydrocele.

Inflammation or Orchitis.—The most common form of this disease is, as you know, connected with urethral inflammation, generally gonorrhœa, and seems, indeed, due immediately to a continuation of the inflammatory process from the urethra along the vas deferens to the testis; and thus it is that the epididymis is so frequently the first part, or that solely affected. The inflammation in these cases generally subsides, and therefore you rarely have an opportunity of examining the anatomical changes which the organ has undergone. In the few cases we have seen there was a mucoid fluid or pus collected in dilated ducts of the epididymis, while the tissue around was rigid and swollen. In the tubes of the testis there was excess of ill-formed epithelium. This condition ultimately leads to induration and contraction of the epididymis. At the same time usually the tunica vaginalis is inflamed and full of fluid, producing a gonorrhœal hydrocele. We have on four occasions met with suppuration of the epididymis in pyæmia; in these cases there was a considerable quantity of pus, running into the condition of abscess. The appearance was then very difficult to distinguish from tubercular inflammation, but the pus was more liquid, and the tissue around more brightly injected; chronic thickening, too, was absent; the disease also did not extend far along the *vas deferens*. Grave inflammation leading even to sloughing has occurred through injuries, as once when the whole length of the cord and testis was laid bare in a run-over case; but in inflammations superficially arising the testis generally escapes; being most liable to those chronic kinds of inflammation due to injury or extension of urethritis. Calculus or gonorrhœa may produce an inflammatory enlargement of the organ, an affection for which testes were frequently excised, and it is probable that some of these older specimens, which are deficient in history, present nothing more than this condition: one, for example, is said to have been growing slowly, in consequence of a blow five years before, but we have not examined it. Sometimes these chronic inflammations end in *suppuration*. The testis becomes adherent, and the scrotum is involved; the abscess then breaks, and the glandular structure becomes exposed, while spongy granulations protrude. This was formerly called *fungus testis*, and was generally removed by the surgeon: specimens of it you see here. Now, however, these cases are viewed as simple inflammation, though there is some doubt whether some of them at least may not originate in a truly tuberculous epididymitis—at any rate, they are cured, except where, by a long inflammatory process, much lymph has become effused into the tissue, and the organ has thus been indurated or destroyed, when excision may be advisable. In such cases as these we have more than once had an opportunity of making a microscopical examination, and we have found remains of the seminiferous tubules enveloped in fibrous tissue, and their natural contents gone.

In most cases of orchitis, however, which come before us after death, the inflammation has long ago passed through all its active stages, and then what you find is a condition which resembles that called cirrhosis in the liver and lung. For the chief part of the section is seen to consist of new fibrous tissue, in which you can find only remains of the natural structure. The testicle is of the usual size, or generally rather smaller; it nearly always adheres to its *tunica vaginalis*. There is then often no history of the disease to be obtained, and we are left infer from the associations what the probable cause of the orchitis may have been. These associations show that it is nearly always syphilitic.

Syphilitic Orchitis—Syphilitic Sarcocele.—The characteristic anatomical feature of syphilitic disease here, as elsewhere, is the "gumma," and this is, as usual, local—indeed, it has no peculiar features in this part, but resembles exactly the gumma found in the liver, &c., forming a yellowish, somewhat elastic knot, which used to be called a tubercle. It is always surrounded by an indurated fibrous zone, and is composed of interstitial inflammatory formation, with more or less evident remains of the natural tubular tissue all undergoing retrogressive changes. The disease is often one sided, like other "tertiary" effects of syphilis. When the gumma is large it is sometimes difficult to distinguish it from a true tuberculous disease; but the following characters will generally guide you. Syphilitic gumma is fleshy or elastic compared with tubercle. It is surrounded by an area of callous fibrous induration. It occurs in the body of the testis, while tubercle affects the epididymis; again, it does not tend to implicate the scrotum and break externally like tubercle, although it almost always causes adhesion between the testicle and its tunic—not invariably, however, for sometimes there is hydrocele. We have seen tubercular and syphilitic disease occur together in the testicle, each with its proper character; but the gumma is not always present in syphilitic orchitis—we have already said that most cases of fibrous wasting of the testis show by their associations a syphilitic origin. Such cases appear as a fibrous infiltration of the organ unequally diffused, affecting some bundles much and others little, so as to cause a mottled appearance of the organ on section, or else the whole organ may be fibrous. In nearly all cases of such fibrous change in the body of the testis you will find other proofs of the presence of tertiary syphilis, so that in every post-mortem on a male subject the testis will offer you convenient and ready indication of syphilitic taint. Though you must remember that, whatever reliance may be placed in the inference from the presence of this fibrous change, you must not set down a case as free from syphilis because the testis has escaped.

Tubercle.—The testis proper sometimes contains a few tubercles in cases of general tuberculosis, though this has been denied. But when the organ is primarily affected the disease shows itself first almost invariably in the epididymis, which is found swollen greatly and converted to a caseous mass, more or less softened and changed to curdy pus, which is contained in spaces loculated with the relics of the walls of the tubes. The exact seat of origin of the disease has been much contested, as a part of the general question whether tuberculous disease belongs to the channels or the interstitial textures of glandular organs, some holding that the process primarily concerns the mucous surface of the tubes, while others think its proper seat is the interstitial tissue. The authority of Virchow is given, as you know, generally to the latter view, and accordingly he holds that it is in the connective substances in and around the tube-walls that the tuberculous matter appears, while he thinks there may be catarrhal products within the tubes secondarily. However this may be, the appearance in an early stage shows the tubes of the epididymis filled with caseous pus, but the walls are also caseous. Around the more advanced disease you often find more or fewer miliary tubercles in the *rete*, or in the testis proper, near the epididymis. On examining these you find evidence that they do arise in the interstitial substance, but whether this was the case in the earlier and now more advanced disease in the epididymis we never could be sure. Such tuberculous abscesses of the epididymis may contract adhesion to the scrotum and open outwards, discharging their contents, while the testis in favorable cases shrivels and may cicatrize, or a fistulous channel may remain, from which exuberant granulations may sprout and deserve the old name of "fungous testis."

The disease always extends along the *vas deferens*, converting this into a thick cord with greyish pellucid walls and a caseous centre. Thus the prostate may be reached, and the tuberculous process may there acquire fresh force, so that caseous prostatic abscesses form and even may lead to perforation outwards or fistulous communication with the bladder. The *vesicula seminalis* of the same side shares in the disease, and thus there is tuberculous disease of the lining of the whole genital system on one side; sometimes the bladder, ureter, and kidney, are also involved. But the testis proper is late in being implicated, and then usually only shows a few miliary granulations, irradiating from the primary seat of disease in the epididymis. Sometimes the miliary tuberculosis arising from tuberculous epididymis becomes general and fatal; of this we have seen several instances.

Morbid Growths.—*Fibroma.*—We have had occasion to observe before that chronic inflammatory products and tumours are not always so easily separable as might be thought. Thus, we just now showed you these specimens of testis in which the enlargement was due to a fibrous element diffused through the tissue. There is some difficulty in knowing whether it be better to call them enlargements from chronic inflammation or fibrous tumours. Generally, however, no such difficulty exists, for if an inflammation can be distinguished from a new growth by the one being an affection implicating intimately the whole tissue, and the other a growth increasing from a centre of its own, then, in many of the cases, the term chronic inflammation or fibrous tumour is easily applied in the respective examples, especially, too, if the tumour occupies a small part of the organ, while the true glandular structure is pushed aside or stretched over it.

You sometimes meet with small polypoid growths of fibrous tissue on the capsule of the testis, especially in cases of hydrocele.

Sarcoma is not frequent in the testis in a pure form; indeed, you must soon be struck with the curious proneness of growths in this organ to show a polymorphous structure. The commingling of several varieties of structural elements in tumours of the testicle makes it difficult to refer the tumours that occur in it to our ordinary classes of morbid growth. Thus, you will find cartilage mixed with medullary sarcoma in a tumour that has numerous cysts in it, and although this is called a compound enchondroma, yet we must not, in so naming it, overlook this curious association of textures not elsewhere found together. Again, you may find mucous tissue, cartilage, glandular acini, and large cysts, together in a recurrent tumour of the testis, and be much at loss to know what class to refer such a growth to. You do meet with testis-tumours of a sarcomatous nature, the texture being of the spindle-celled variety; but most commonly there are cysts present and the cells have communicating processes which make a meshwork in which a mucous fluid is contained, so composing cystic myxosarcoma.

Cystic myxosarcoma or "cystic testicle" is a name given to tumours of the testis that are chiefly composed of cysts. The solid part of their structure varies much; they grow to a large size. In these specimens which are characteristic examples we found the stroma to resemble the tissue of which nasal polypi are composed; that is, a lax "fibro-cellular" (myxosarcomatous) web, made up of spindle-formed and multipolar cells connected by a filamentous meshwork in which is a submucous fluid. There were, however, in some parts numbers of free lymphoid or epithelioid cells, while in other parts the texture was condensed to an almost purely fibrous structure. The cysts of various sizes contained either serum or mucous or colloid fluid. Such tumours generally are "benignant," and do not recur on removal. But you must know that all cystic tumours of the testis are not so simple; some, curiously, contain muscular fibre, striated or non-striated, or cartilage, or else they may contain cancerous tissue or all these together, and then prove malignant, recurring, as in this instance, after removal. As to the origin of the cysts themselves, some are probably new formations, others are dilated tubules. Sir J. Paget in a remarkable case of enchondroma, in which the cartilage grew in cystic spaces, found that these spaces were dilated lymphatics.

Myxoma occasionally is found as a large, soft, mucous tumour of the testicle, described by Lebert as fibro-colloid.

Carcinoma.—Cancer varies in different parts of the body according to the texture in which it occurs; and thus, although there is such a form as medullary with only a very tender matrix, and scirrhous with a firm and abundant one, yet the differences of these are due in great measure to the character of the organ in which the disease occurs; thus, in the dense fibrous structure of the breast, cancer is scirrhous, while in the testis it is *medullary*. You see many examples of this disease on the table; the organ is much enlarged, and, when cut, is found to consist of this soft matter, which is well called encephaloid, for it very closely resembles the structure of the brain. Remains of the testis will generally be found on the surface, having been pushed away by the increasing growth; and you often find, as you see here, yellow deposits of degenerating fibrin as well as these yellowish-white masses of the decaying cancer itself. Cysts occur in carcinomatous testes, and may be so much developed as to reduce the carcinoma to the relation of an interstitial material. Sometimes cartilage is present in these tumours. They expand the tunica albuginea, and often set up secondary tumours of the iliac and lumbar glands.

Scirrhus or hard carcinoma is rare in the testis, but a few well-marked cases have occurred. Here is one which was removed by Mr Bryant. The structure is characteristic.

Enchondroma—This is a very remarkable, though not uncommon, disease of the testis; a very good specimen of it you have here. You see the organ is enlarged, and in it are these translucent nodules which under the microscope had the appearance you see in this drawing, namely, that of well-formed cartilage. But you will observe that this cartilage does not form a continuous mass; it appears as a number of distinct pieces, separated from each other by an intervening substance of fibro-cellular character, and thus it is very different from the pure enchondroma so common in the bones. The origin of cartilage in the testis is very curious; it is a good example of *heteroplastic* growth as defined by Virchow; that is, of the rise of a tissue widely different in character from the matrix in which it takes origin. Also the enchondromatous testis well illustrates Virchow's teaching that such heteroplastic origin brings with it malignancy, for while cartilaginous tumours on the bones—natural seats of cartilage—are generally innocent, a great suspicion surrounds the character of any cartilaginous tumour of the testicle. In a case which occurred to Sir J. Paget, even of pure enchondroma of the testis, the cartilage returned as a secondary growth in the lymphatic glands and in the lungs. We have here a small portion of the lung which is consolidated by it. The cartilage is, however, very rarely pure, but rather, as in the case we first showed you, it is combined with other morbid material, either myxomatous, sarcomatous, or even not unfrequently carcinomatous. In the return of such tumours after removal cartilage generally grows n in the new mass, which appears usually in the iliac glands or in the viscera, although we have known such a compound cartilaginous growth return in the scrotum. One interesting particular in these growths is the development of the cartilage within hollow spaces, cystic or tubular, in which nodules and rods of cartilage lie apparently free. Paget found these spaces to be dilated lymphatics, and traced the cartilage up the lymphatics of the spermatic cord. Virchow saw the cartilaginous growths both in the blood-vessels and lymphatics. Quekett described them as in the ducts. We have found them in lymphatic spaces, while some dilated ducts contained curiously formed compact masses of epithelium very like fœtal cartilage. The growths in the lymphatics at first appear as if they were free, but closer examination shows them to be connected with the vessel's wall, from which they grow as polypoid masses, projecting into the dilated channel.

Wherever cartilage is formed you are likely to have osseous tissue, and thus you might not unreasonably expect to meet sometimes with bone in these diseased testes. In the lower animals, we believe, complete ossification of the testis sometimes takes place.

TUNICA VAGINALIS

Inflammation.—Inflammation of the tunica vaginalis with production of lymph is generally artificial, caused by injection in order to obliterate the cavity after tapping for hydrocele. If by chance you should get an opportunity of seeing the membrane a few days after the operation you will find it showing, very well, the usual characters of acute serous inflammation; the cavity in a case we inspected was filled with soft yellow lymph, and the surface of the testis covered with a firmer layer. Such inflammation is very rare as a primary disease.

Hydrocele, or collection of serum in the tunica vaginalis, must be ascribed to a chronic inflammation of that membrane. In cases of great anasarca, even when the scrotum is largely implicated, the sac of the tunic is almost free from fluid, so that we cannot regard hydrocele as dropsical. Again, the disease resembles in character a chronic pleuritic effusion, and when it is tapped we find that the fluid from it coagulates. Sometimes the sac communicates with the peritoneal cavity, and then the hydrocele is called *congenital.* This differs obviously from the true closed hydrocele; the fluid may only descend from the abdominal cavity.

The tunica vaginalis generally may be found thin and pellucid, as you know; but if the case is of long-standing, and the part has been subjected to much irritation or injury, as is often the case, you find in it the results of chronic inflammation, so that the serous membrane has become very much, and sometimes enormously, *thickened;* in some of these cases it is a question whether the inflammation may not have been due to curative measures formerly adopted, for we do not think the serous cavity is always obliterated by injection. The sac may be greatly thickened, while the testis is reduced to a small size by pressure of the fluid. The thickening of the sac is often not uniform. Sometimes there are merely indurated spots on, or hard *nodular bodies* projecting from, its inner surface or that of the testis; these occasionally become *cretaceous*, and, indeed, the whole membrane may become bony or calcified. The depending fibrous bodies which form on the testis may sometimes drop off, as occurs in the joints, and may be found loose in the tunica vaginalis. In one or two such which we have examined the texture was not true bone.

Hæmatocele.—This name is given when the tunica vaginalis becomes filled with blood, a state generally arising from injury. The blood usually becomes absorbed, and no injurious result follows; sometimes the fluid parts are taken up, and the fibrin forms distinct layers, as you see here, resembling the interior of an aneurism; in the more unfavorable cases the blood softens down, and a sanguineous cyst is produced; in such a case, if the cavity be tapped, a fluid is drawn off corresponding to changed blood in other parts—viz., chocolate-coloured fluid, containing numerous plates of cholesterine and hæmatoidin crystals, as we have before described. Very often a hæmatocele is produced by injury to a hydrocele, either in tapping, or by accidental blows, or inflammatory rupture of vessels.

Spermatocele—Encysted Hydrocele.—Sometimes cysts form at the upper part of the testicle about the epididymis. One or more of them may grow into a large size and thus closely resemble common hydrocele of the tunica vaginalis, hence it is called *encysted hydrocele.* The fluid drawn from them is then apt to be milky from admixture of semen, whence the name spermatocele used for such cysts. The exact origin of the cysts is a subject of great interest; much light has been thrown upon it, yet some discrepancy of views still remains; but they are now almost universally regarded as diseased enlargements of the remains of the Wolffian body. A part of this body you know was converted into the epididymis, while its duct formed the *vas deferens;* but some parts of the body remained unused. It is thought by Virchow that such remainders, being still in connection with the epididymis which arose from parts contiguous with them, may enlarge and form cysts into which the spermatic fluid regurgitates, perhaps through obstruction of the *vas deferens.* Paget suggested that the cysts were new growths and produced semen in virtue of their nearness to the testes. Curling argued against this view that semen is not found in the cysts when small, as would be expected, but when very large; and we know that large cysts in glands generally cease secreting even when they arise by dilatation of the proper secreting sacs of the gland. The fact that semen only appears in the larger cysts is equally against Virchow's views. We too have found that the semen is not present in the small cysts, and hence believe, with Curling, that it probably enters the large cysts during their growth through opening of the ducts by their pressure, just as bile enters an hydatid cyst.

The cysts may be more or less numerous, sometimes a single cyst is embedded in the epididymis; they are lined by a beautiful squamous epithelium very separable from their wall, and they arise in the subserous tissue of the epididymis, or of the neighbouring part of the tunica vaginalis, quite unconnected with the epididymis. The mass of cysts may make a considerable tumour above the testicle.

VAS DEFERENS AND SPERMATIC CORD

Hydrocele of the Cord, a collection of fluid in an abnormally persistent portion of the *processus vaginalis,* sometimes occurs as a distinct form of disease, as you see in this specimen; the inflammatory changes which we have described may then occur in the serous membrane, producing thickening or even earthy degeneration, as in this case.

Encysted hydrocele of the cord is a distension of the areolar tissue around the cord with fluid. (Some call this hydrocele of the cord, and give the title "hydrocele of the processus vaginalis" to what we have called by the former name.)

Varicocele is a term given to a varicose condition of the spermatic veins, which form a large tortuous bunch at the lower part of the testis; sometimes you find thrombus in a varicocele. Sometimes the vessels become ossified, and we have seen them forming a hard mass, associated with a similar cretification of the iliac and pelvic veins. The *vas deferens* may also sometimes be found ossified.

Tubercle may extend along the cord, when the same form of disease exists in the testis.

Cancer, in the same manner, may involve the cord, and in this specimen you see a *melanotic tumour.*

VESICULÆ SEMINALES

Atrophy of these organs occurs often in old age, and the enlargement of the cells, which is often seen in the aged, is probably a species of degeneration.

Inflammation and Suppuration is not unfrequently met with in cases of pericystitis and abscess of the prostate, where the base of the bladder is involved.

Tubercle.—In cases of tuberculosis of the testis and spermatic cord, the vesiculæ seminales may be affected, as you see in these specimens; often only one is involved, when the disease of the testis is unilateral.

Carcinoma, when affecting the neighbouring organs, may involve also the seminal vesicles. In one example reticular carcinoma arose from the vesicles themselves, extending into the tissues around.

Concretions, as you see here, may also be met with in them.

PROSTATE

Hypertrophy.—This is the most common and important disease of this body, especially when it leads to outgrowth of a portion of the organ into the bladder, constituting what is called the middle lobe. The prostate may be uniformly enlarged, but such a uniform hypertrophy of the whole organ is rare; for you will nearly always find on section of an enlarged prostate that the increase is due to a formation of one or more circumscribed tumours in its substance, and this even when on the exterior the organ appears to be equably augmented. The tumours are of the same constitution as the prostate itself. This you know is composed of glandular tissue, sparingly present in a fibromuscular mass which acts as sphincter to the bladder. According to Virchow you may find the glandular, the fibrous, or the muscular matters to predominate in the new growth, while the general character is maintained. This is true, and is a matter of pathological interest; sometimes, indeed, you find a small quantity of glandular product in sections of such tumours. But yet the main run of cases show among themselves very much the same kind of material, and this is very like that of fibroid tumour of the uterus. When you recollect how close are the homological relations of the uterus and prostate this must arrest your interest, although it is not quite true that the prostate is in reality the masculine equivalent of an uterus. For homology to the uterus is strictly limited to the *vesicula prostatica*, yet, certainly, the behaviour of the uterus and prostate as to these myomato growths is curiously alike. In each organ they come as distinct tumours in declining life, and they have in the two almost identical characters. The favourite seat of them is in the thicker part of the base of the prostate at the neck of the bladder behind the urethra, where, rising up, they form valve-like masses that too effectually close the bladder, so that at last there is no way out of the cavity except by tunnelling the prostatic lobe with the catheter; such a mass is often called a "large middle lobe," but it is really a new growth, and may be found separate from the main mass of the organ; these enlargements are rare in the upper part of the prostate. The effect on the prostatic part of the urethra is to flatten its channel, while at the same time the extent of the urethral surface is greatly increased, so that, for instance, you may find the canal three inches around, but flattened closely. The channel may be bent out of its ordinary course, but its great width prevents any real stricture as a consequence of this deviation.

Although such enlargements probably occur more frequently in advanced years than in the middle period of life, yet they by no means are necessarily connected with age: for many years past we have been in the habit of examining the prostate in old persons, and have found that, speaking generally, its enlargement only occurs in those cases where it produces symptoms and has been a source of complaint during life. We can, therefore, quite confirm the statement of Sir Henry Thompson, that enlargement of this organ is not a necessary attendant on old age; indeed, we have found it sometimes rather atrophied. We do not mean that this enlargement is not more frequent in advancing age, but it is not one of its necessary or ordinary concomitants, as grey hair or calcified blood-vessels, though this was formerly thought to be the case.

Fibroid Degeneration. — So far from enlargement necessarily occurring in old age, we have sometimes found the organ atrophied. Also in younger people we have met once or twice with a condition which might be called fibroid degeneration; and in one case this was associated with a similar affection of the testis, which, we have no doubt, was due to syphilitic cachexia. The organ was converted into an amorphous hard substance, interspersed with opaque white parts indicating a fatty and granular degeneration.

Inflammation and Abscess.—This is a not uncommon affection, coming before the surgeon either as an acute inflammation caused by gonorrhœa or injuries, or else as an old disease in connection with stricture, when it is frequently found in the post-mortem room. In long-standing cases of stricture, where, perhaps, urinary fistulæ have long existed, and especially where false passages are present, and the patient has died from some acute mischief about the part, either with or without external extravasation of urine, the prostate and coats of the bladder are often found involved in a suppurative process. There may be fistulous connection of the urethra with the bladder or rectum, or an extension of the fistulæ under the peritoneum, or into the perinæum. It is difficult to say, when such mischief has occurred, what has immediately set it up—whether it be a mere continuation of the urethritis backwards, or the effect of injury done to the prostate by catheters, but we find openings on either side passing into this organ; and when the lobes are divided they are found full of pus, or the whole structure, perhaps, is in a sloughy condition. We are under the impression that practitioners are scarcely aware how, in their very bad or fatal cases of stricture, the mischief is often deep seated, and that the prostate, the bladder, and the pelvic cellular tissue, frequently share in disease of the urethra, which shows itself superficially in external extravasation.

You sometimes find the prostatic urethra widened much in cases of stricture by the pressure of the urine. In this case it is sacculated on each side, apparently from the same cause.

We may here show you a specimen of prostate, which we removed from a man who had been operated on for lithotomy fourteen years before, and in which you see not the slightest trace of the incision.

Tubercle.—This is not usually a primary disease, but merely a part of a general tuberculosis of the urinary or genital organs; but the prostate may be extensively infiltrated, and the tuberculous mass may soften down into large cavities, which become a source of much suffering to the patient.

Cancer of the prostate is very rare, but it does sometimes occur as a primary disease. Several cases have come under our observation, once of soft, twice of firm reticular carcinoma. The disease is not generally limited to the prostate, but has a remarkable tendency to extend to the tissue around.

You sometimes meet with simple *cysts* in the prostate; they are rare. Here is one opening into the urethra.

Concretions or Calculi.—These may be found, on making a section of the prostate, as small yellow translucent bodies like amber, or sometimes as blackish grains; when large they are facetted by mutual apposition and friction, like gall-stones. This would distinguish such concretions from any other kind of calculi from the urinary passages. Sometimes, however, large calculi form in the organ, as you see here, where also they are endeavouring to escape into the urethra.

PENIS AND SCROTUM

Malformation.—Some important malformations of these organs are connected with extroversion of the bladder. In *Hypospadias* the urethra remains open on its lower surface for a greater or less extent, along with a proportionate degree of smallness and general incompleteness of the parts. In its extreme degree the urethra is open all along the scrotum, which is thus divided into two halves, while the testes, of small size, are retained more or less above their proper position.

Inflammation.—The surface of the glans is liable to simple and gonorrhœal inflammation, or *Balanitis*, producing pus between it and the prepuce; if the latter be closed by Phymosis the matter may collect and cause ulceration, and perhaps perforation, or even gangrene of the prepuce.

Sometimes the body of the penis inflames, and the *corpora cavernosa* are found to contain lymph or pus. This is not frequent; it may result occasionally from injuries or severe gonorrhœa, or it may arise as in other exposed parts through putrid fevers. The effect may be general pyæmia; or destruction of the part; or fibrous wasting. Occasionally calcareous matter has been known to be deposited in old fibrous patches. Sometimes in old dropsy cases you will find pus in the scrotum.

The terrible inflammation of the external genitalia produced by extravasation of urine will soon become familiar to you in the wards, and you have no doubt studied in the dissecting room the means by which the fluid is prevented from descending upon the thighs, through the union of the superficial fascia with Poupart's ligament, and the pubic arch.

Erysipelas.—We would draw your attention to several cases in which we have found all the external characters of extravasation of urine in the form of *gangrenous* erysipelatous inflammation in this region, but without any disease or injury of the urethra at all, so that the disease proved to be a *simple erysipelas* of those parts.

Syphilitic inflammation.—It is not often that you meet with open chancres post mortem. The peculiarities of syphilitic chancre, their greyish indolent surface, and congested callous margin, all belong to the living condition. The induration alone remains after death. This induration, you know, may persist some time after the healing of the sore. The material causing the induration is composed of roundish cells similar to those that make up the substance of granulations in healing wounds, so that Virchow classes it with "Granuloma." You should accustom yourselves to be quick in recognising scars of bygone syphilitic sores. Should a scar of chancre be present, always look for the scar of a bubo. The presence of the latter takes away nearly all the weight of the former as evidence of syphilis, since, as you know, chancres that cause suppurating buboes *very rarely* cause syphilis.

Eczema, herpes, boils, &c., occur on the penis as elsewhere on the skin.

Condylomata are frequently found on the external genitalia, both the *acuminated* form, due to simple or gonorrhœal irritation, and the *broad* form, which is truly syphilitic. Condylomata of the first kind arise in, and take their character from, the papillary layer of the corium, while those of the second consist of the deeper skin infiltrated and raised into elevations by the same granulation-like material which is found in the base of the hard chancre, and which Virchow calls granuloma.

Epithelioma.—This generally assumes a warty character, and thus very large "cauliflower excrescences" are produced on the organ. Such a term is peculiarly applicable to a fine specimen like this; while in this other one you see the disease at its earlier stage, where merely the adjacent parts of the glans and prepuce are affected. The structure shows very plainly all the characters of epithelial cancer. We do not know that surgeons have yet agreed as to its greater frequency in those who have been the subjects of congenital phymosis, but we can well believe how any unnatural condition should excite the disease, and especially where any irritating secretions are retained. Like epithelioma elsewhere, this is a local affection, and when removed early and with sufficient margin, does not return. If it be allowed to continue, the disease involves the glands. On examining the body, you may find that the more superficial have contaminated some of the deeper glands, and a few around the iliac vessels may be implicated, but, apart from this, no disease will be found in the body.

Carcinoma.—You not unfrequently meet with well-marked carcinoma of the penis; we have examined several specimens. The structure of the growth has generally been markedly of an adenoid type, the pseudacini being often very well formed, but the cells were large, multiform, and with large bright polynucleolated nuclei.

Cancer, or epithelioma of the scrotum, formerly called chimney-sweepers' cancer, is the same kind of disease as that just mentioned, but affecting the skin of the scrotum: it is caused especially by the irritation of soot, and is now, we believe, much less frequent than formerly; a round raised raw surface is seen on the scrotum, having an ichorous discharge; when excised early a perfect cure results; when not, the inguinal glands become fatally affected in the same manner as we have just mentioned. But very different is the result of carcinoma of the testis, which, being of the purely medullary kind, very soon involves the lumbar glands, and the patient dies with large tumours in abdominal and other organs.

Chronic Hypertrophy of the Skin and Cellular Tissue, or Eleph tiasis Scroti.—This is the same kind of disorder which occurs in leg under the designation elephas, or elephantiasis Arabum. It consi of a simple hypertrophy of the tegumentary tissues. It may aff chiefly the penis or the scrotum, but generally reaches its larg development in the latter, dragging the integument and prepuce do so as to produce distressing impediment to micturition. You may by our Chinese pictures to what immense size these tumours grow the East; this is partly due to the want of surgical skill to rem them when small. One of the most remarkable is that in the museu which was removed by Mr. Key, from Hoo Loo, a Chinaman; it wei several pounds. A disease alike in kind, but very different in degr is frequent enough in this country.

In some cases, however, when the scrotal tissues were swollen, as elephantiasis, there were found sarcomatous elements mixed with hypertrophied connective substance.

The teguments of the scrotum have been found distended decaying blood-clot, so as to form a tumour.

We have already shown you a scrotum containing *chalky deposit* a gouty subject.

DISEASES OF THE FEMALE SEXUAL ORGANS

VULVA

The extreme degrees of malformation in which the external genitalia are quite absent or very imperfect are not of practical interest, as they occur in unviable fœtuses only.

Malformation.—We have seen that in hermaphrodites the genitalia may resemble female organs as to external conformation, while the individual is essentially male in the possession of testes. So likewise you find that when the individual is essentially female in the possession of ovaries the external organs of generation may approach in superficial characters the appearance of the male. Thus the clitoris may be long, and pierced by the urethra, while the vagina becomes small and the uterus and ovaries imperfect, the latter, perhaps, descending into the labia.

The labia or nymphæ may grow to too great a size, or may be united together.

Inflammation.—Skin diseases become localised about the vulva; thus eczema, herpes, erythema, erysipelas, &c., are obstinate there; also lupus may appear in its several forms. *Noma* or gangrenous phlegmon, which we have described as it appears in the cheek, is also found here, more frequently in children.

Injuries may arise during a severe labour, owing to pressure of the child's head, by which a large vessel, generally a vein, is lacerated; blood is effused, and a great tumour is formed; if this be opened, altered blood is discharged. You may see similar swellings in the surgical wards, arising from direct blows, and they occasionally appear to have been caused by strains. Such swellings may suppurate and even cause death by extension of the inflammation to the interior of the pelvis.

Œdema may be a result of pressure during pregnancy, or may arise in general dropsy, being in some cases excessive when the dropsy is slight elsewhere. When œdema becomes extreme the part may inflame and suppurate, and there is danger of gangrene. A *gangrenous* condition may also be set up during putrid fevers.

Morbid Growths.—*Condylomata*, both acuminated and broad, such as we have described in the male genitalia, are constantly met with also about the vulva. The former, due to simple irritations or gonorrhœa, may be of a simple kind, like those which occur on the hands, though they often form large masses; the latter are syphilitic, they too may grow to a large size. You also meet with conditions corresponding to the elephantiasis scroti of the male, consisting indeed of similar hypertrophy of the skin and cellular tissue. This form of disease may here put on the shape of a polypoid outgrowth, or may have a broader base and be continuous with the labium. Here you see a large tumour from the labia, and another from the clitoris.

Epithelioma, or epithelial cancer, sometimes attacks these parts, as represented in this drawing; but it is not a very common affection. It shows the usual characters of this disease.

Fibrous tumours occur in the vulva as in other parts; they are removed by the surgeon, as you may frequently see. They are composed of dense cellular tissue like the labium itself, and may be present in any part of the vulva, sometimes growing to a great size. They differ from elephantiasis in being circumscribed, and not indefinitely continuous with the general subcutaneous textures.

Lymphomatous tumours also occur here occasionally, sometimes combined with *fibroma*.

Encysted tumours may also sometimes be met with here; we have seen several such, and they contained a limpid, watery, or a mucous fluid. Dermoid cysts sometimes appear also. We have already spoken of blood-cysts and abscesses.

Hernia.—We may just allude to tumour in the labium produced by inguinal hernia.

VAGINA

Malformation.—In some cases of imperforate anus the rectum opens into the vagina. Sometimes the vagina is double, as in this case, where the uterus is double also; a double uterus generally indeed accompanies this state, but sometimes the vagina is nearly divided when the uterus is single. Again, besides sharing in the faults in the direction of hermaphroditism, in which the vulva is chiefly concerned, the vagina may be absent, either when the uterus is also absent, or in the presence of the uterus. The whole canal may be closed up, or only its upper or lower part, or both. Sometimes such congenital occlusion of the vagina seems to be the result of inflammation during fœtal life, for it is found when the development of other parts shows no failure. It then resembles the results of severe ulcerative inflammation in early life, which likewise may produce almost or entirely complete occlusion of the passage.

Sometimes the vagina is closed by an *imperforate hymen*, when an operation may have to be performed for retention of menses, and a large quantity of dark pitchy fluid, like treacle, consisting of altered blood mixed with mucus, may be drawn off. This operation is attended with great danger through the risk of escape of menstrual fluid out of the Fallopian tubes into the peritoneum, indeed the proportion of deaths is very great. This is, we think, especially the case when the occlusion of the vagina is more extensive, through congenital malformation or through ulceration, as in these cases there is more impediment to escape of the fluid, thus increasing the danger of extravasation of menses into the abdomen.

Variations in Size.—*Prolapsus, &c.*—Dilatation occurs above the obstruction in the cases of retained menses above described, or through prolapsus uteri, &c. When there is dilatation or loss of tone, as after parturition, the vagina itself may prolapse either around all its circumference, like prolapsus ani, or more frequently by its anterior or posterior wall. When the anterior wall comes down the bladder may be brought with it, forming a *cystocele;* or the rectum when the posterior wall descends. Sometimes the small intestines in Douglas's pouch force in the back of the tube, and protrude into the vagina or at the vulva, forming an *intestinal vaginal hernia*. The uterus descends somewhat in any of these protrusions of the vagina.

Contraction of the vagina is caused by pressure outside it or by cysts, &c., in its wall, or through cicatrices of ulceration within. The tube may be lengthened and narrowed by the dragging up of the uterus adherent to a tumour growing out of the pelvis.

Inflammation.—Vaginitis shows itself generally as a catarrh, which is usually gonorrhœal. But there are other idiopathic forms of *vaginitis* which correspond to the varieties of stomatitis; thus, in children out-patients, an inflammation accompanied by a discharge is very frequently seen, which is often very difficult to distinguish from gonorrhœa. Also *diphtheritic* or pellicular inflammation is found in the vagina, generally in severe febrile exhaustion after parturition, or in putrid fevers, or in diphtheria itself; *ulceration* or *gangrene* may occur beneath the membranous layer; syphilitic ulceration we need scarcely mention. We meet too here, as in the mouth, with a rapid *gangrene* or *sloughing*. This occurs in the external genitalia of children, and the same name, *noma*, has been given to it; it has sometimes appeared as an endemic in particular localities, when its nature cannot be mistaken; but, meeting with an isolated case oment believe it to have been the result of violence. You must be quite prepared for any such suggestion, and remember that, amongst the poor, where there is often a common sleeping-room for many individuals, a charge of violence is very apt to be made; but in these cases of idiopathic inflammation it is rather the vulva and external parts than the vagina which is affected. A case which occurred here a few years ago might have given considerable difficulty in diagnosis had there been any suspicion of violence; for not only were the labia and adjacent parts mortified, but owing to the purpuric condition of the body, which had led to the disease, large livid marks, exactly resembling bruises, existed on the arms and legs. Like cancrum oris, it is generally a consequence of measles, or one of the exanthemata. Gangrene may of course arise from direct injury or during labour.

Aphthous inflammation sometimes occurs in the vagina and vulva, as in the mouth, and it forms a very troublesome affection in women. The mucous membrane is inflamed, red, and covered with small ulcers.

We need scarcely allude to *leucorrhœa*, except to remind you of the advantage sometimes gained by examining these discharges by the microscope; sometimes the knowledge so obtained may be of great service. The white discharge from the vagina contains merely mucous corpuscles and more or less perfectly formed pavement epithelium, while that from the os is more gelatinous, and if it has escaped from the cervix may contain cylinder epithelium.

Morbid Growths.—*Warts or warty tumours* are sometimes met with in the vagina, as you will observe in our wax models.

Myoma.—Tumours just like in structure to the ordinary uterine fibroid occasionally but rarely form in the vagina, especially the posterior wall. Sometimes when in the upper part, they have arisen originally in continuity with the uterine fibres, but detached themselves afterwards during growth, in the same way as many subperitoneal uterine fibroids do. Such tumours may project in a polypoid manner.

Mucous polypi grow sometimes from the walls of the vagina. In a case we not long ago examined, where a uterine polypus existed, another was found growing from the vagina; and in another case, a polypus which had been removed by ligature sprang altogether from the vagina; it was as soft and vascular as the ordinary uterine polypi. These are not very common.

Cancer is unusual as a primary disease; but in cases of cancer of the uterus the upper part of the canal is generally involved. In the rarer form of disease, *epithelioma* of the external parts, the lower end may be involved.

Cysts, like this one, are sometimes removed from the vaginal walls.

Injuries of the vagina are not unfrequent as consequences of pressure of instruments, &c., during labour, when the perineum, rectum, or bladder may be torn or so bruised as to subsequently suppurate and slough at the spots affected, causing fistulous openings. Clumsy attempts to apply the forceps may lead to perforation of the vagina into the peritoneum. The passage may be wounded by piercing instruments. The breaking of glass syringes may cause severe and even fatal wounds in it.

Fistular communications with the bladder, ureters, or rectum, arise through the above-mentioned injuries or else through ulceration. Here is a specimen of recto-vaginal fistula caused by syphilitic ulceration; it is contracting, and such fistulæ may heal spontaneously. But vesico-vaginal fistulæ being constantly irritated by the passage of urine are not cured without a plastic operation.

UTERUS

Malformation.—This organ may be altogether *absent*, as we have now seen on two or three occasions. This may happen when the ovaries, &c., are well developed. On dissection the merest trace of the fundus, as a slight ridge, may be seen on the posterior surface of the bladder, or it may be quite absent; the organ may be found only in the form of a small sac or small solid mass, or to the small uterus dilated cornua may be appended; if the ovaries are present, the characters of the female sex appear in the general conformation. Or the uterus may be double, as you see in this specimen, where also there are two perfect vaginæ; or you may see a disposition to duplicity, which shows itself in the uterus being bicornuate, that is, the fundus separating into two distinct cavities. Sometimes one side of the uterus is ill-developed or scarcely developed at all, while the other is well developed. This forms the "unicorn" uterus. The divided or the unicorn uterus may produce children, nay, in cases of divided uterus it appears that pregnancy may start on both sides at different times.

To understand the doubleness and the unicorn condition of the uterus you must remember that in the early months of fœtal life the uterus is formed in two distinct symmetrical parts, which subsequently unite and are moulded together to form the single body of the organ. One side fails in the unicorn uterus, and the two sides continue too long separate in the double uterus. The doubleness differs in degree; sometimes it is only at the fundus, or it affects the body, or the neck is implicated. Sometimes through excess of development the cavity is divided by a septum when there is no sign of this outside.

An abnormal figure may be given to this organ from various tumours, &c., compressing it, but especially is it seen altered in cases of ovarian disease, where adhesion takes place and the organ is forcibly drawn upwards; by this means the cervix becomes immensely lengthened. It is curious to see how deeply it is often imprinted by scybala after death, so deeply that no doubt can be entertained of its often being so imprinted during life.

We may here just remind you that you may sometimes wish to know, when you open a body, whether the uterus has ever been impregnated, or borne children, or whether it be a *virgin uterus*, as the organ is called which has never held an ovum. The former is somewhat larger than the latter, and the os bears traces of having been dilated, or even lacerated, having an irregular puckered look, as compared with the smooth oval opening of the virgin uterus; the cervix is not so long as in the virgin organ, and the rugæ are in great measure obliterated; and the cavity has a rounded form instead of a triangular shape.

Malposition.—*Prolapsus uteri.*—The uterus may descend into the vagina so as to protrude externally and draw down with it the bladder under the pubic arch, compressing the ureters against the arch. We have known on two occasions compression of the ureters from this cause to produce fatal pyelitis. It is comparatively rare for the whole body of the uterus to descend; if it does it generally occurs after parturition, but the uterus may be drawn down by the causes that induce prolapsus of the vagina. What is more common is a prolapsus of the neck of the organ; the body may then descend a little way, but the neck lengthens out enormously, while its lips are somewhat wasted, and the whole may be protruded external to the vulva. The overgrown mass has the general structure of the neck of the uterus, but is more congested; its mucous membrane is generally inflamed, abraded, or ulcerated. The mucous follicles may be enlarged. Sometimes such hypertrophied necks are removed from the uterus by operation, but great care must be taken to avoid opening the peritoneum. Some distinguish as a form of prolapsus a mere elongation of the intravaginal portion of the neck; this is, however, a comparatively unimportant condition.

The uterus may be *anteverted*, or *retroverted*, or *lateroverted*; the whole body being turned forwards, backwards, or to one side. These conditions, especially anteversion, are very common in lesser degrees, for the organ is very moveable; they are commonly quite innocent, but they sometimes form a part of the cause of uterine annoyance, especially when the version is complete, so that the fundus of the uterus is downwards and its neck upwards. The bladder, rectum, or ureters may then suffer considerably so as to give rise to serious results. Nevertheless, it is remarkable that these versions are so frequently present when no complaint whatever has been made, and we ourselves have never happened to meet with any case where versions of the uterus led to diseased conditions discoverable post-mortem. Sometimes the organ is put aside by pressure or traction of tumours or adhesions; here, however, the displacement is evidently of secondary importance.

You sometimes find in the dead body the uterus bent on itself at the os internum, or junction of the cervix with the body; it may either bend forwards and, forming a right angle, rest on the bladder, or, being bent backwards, rest on the rectum; the one condition is called *anteflexion*, and the other *retroflexion;* the former being the more common.

Whatever troubles are traced to these conditions during life, and they are many, they always appear very innocent on the post-mortem table. The same conclusion would follow from M. Panas' observations. It appears that among 114 persons, not suffering from any uterine disorder, 70 had various versions and flexions of the uterus; 40 anteflexions; 12 anteversions; 3 retroflexions; 3 retroversions; and 12 lateroversions.

The uterus may be drawn up very high into the cavity of the abdomen, when it is fixed to ovarian tumours, &c., the vagina then being elongated and conical in form. Rokitansky says that great fibrous tumours may in their growth tear the body of the organ from its neck.

Inversion of the uterus happens after labour; it appears as a red mass projecting into the vagina, and it is very difficult to distinguish from polypus during life, but the absence of the uterine cavity and the sensibility of the surface will generally guide you. It may remain for a long time before its return is effected, and some danger is incurred in rough attempts at its reduction. Here is an antero-posterior section through an inverted uterus. You will see how completely the organ is turned inside out into the vagina through the os uteri. The Fallopian tubes and ovaries are drawn into the peritoneal hollow, which makes the inverted cavity of the organ. Death was caused by pyelitis, set up through compression of the ureters by the tumour against the pubic arch. The inversion is not always so complete as in this specimen; the body may be inverted into the neck, or the fundus into the body. Partial inversions are sometimes caused by the traction of polypi within the cavity.

Congestion and Hæmorrhage.—In all diseases where the blood disposed to stagnate—whether from mechanical cause, as in cordis, or from the unhealthy state of the blood itself, as in fevers—you may often find the uterus and ovaries, like other organs, highly congested, and perhaps in a state of catarrhal inflammation; and then, just as there may be a flow of blood from the intestine or other surface, so also there may be from the uterus. You may thus find in fever and other diseases the interior of the uterus containing blood, and the question of menstruation will be suggested to your mind, as it constantly is to the nurses during life, although nothing but a morbid congestion exists. Growths within the uterus lead to intense congestion, and perhaps to hæmorrhage, either by oozing from or ulceration of their surface. You may find a passive form of hæmorrhage into the wall and cavity of the uterus in old age.

Hæmorrhage about the Uterus.—*Retro-uterine hæmatocele.*—This name is given to a lodgment of blood in Douglas' pouch. Blood might be expected to be frequently present there, since this pouch is the lowest part of the abdominal cavity; so that it naturally receives the products of any peritoneal hæmorrhage. We have seen some cases in which blood had collected there from such sources, as the following: cirrhosis of the liver and heart disease, which had caused intra-peritoneal bleeding, though a very rare consequence in those conditions, extra-uterine fœtation, inflammatory congestion of the ovaries from the neighbourhood of ovarian abscess or suppurating ovarian cyst, escape of retained menstrual fluid in imperforate vagina, rupture of a varicose vein in the uterus, operations or injuries about the abdomen, ruptured aneurism; in short, every source of intra-peritoneal hæmorrhage. But although any mass of blood so caused is generally adopted as a retro-uterine hæmatocele, something more special to the genital system is usually signified by the term as used by "gynækologists." In particular, a regard is had to its assumed occurrence through escape of menses into the peritoneum, or some other incident causing bleeding during menstrual congestion, or through hæmorrhage from an ovary with its pedicle twisted. There is still much dispute as to the pathology of the tumour-like masses assumed to be blood and found during life behind the uterus, some observers thinking that such menstrual accidents as regurgitation of the menses or rupture of a vein may induce such hæmorrhage in healthy individuals; coagulation and subinflammation causing the blood to form a semi-solid mass. They appeal chiefly to the history of the attack, which often comes as pain and shock in the early days of menstruation. On the other hand, it is contended that you cannot have a tumour from free blood, and that when the blood is encapsulated, as it is found post-mortem, there is generally a good deal of disease present, which, probably, may have preceded the bleeding, so that the latter arose from the new vessels of inflammatory false membrane. Here comes in, indeed, the whole question concerning the primary or secondary origin of bleeding and inflammation in serous sacs, which we have considered in the history of hæmatoma of the dura mater, the authority of Virchow bearing in favour of the secondary nature of the bleeding in both cases alike. We have mentioned the rupture of a varicose uterine vein into the peritoneal cavity; this case gives support to the observations of rupture of varicose veins in the broad ligament, &c., collected by Bernutz and Goupil. In the few other examples we have seen, which could be at all referred to proper genital hæmatoceles, it appeared that old disease preceded the hæmorrhage.

Hæmorrhage into the broad ligament has been described under the name of *peri-uterine hæmatocele*.

Inflammation.—Acute metritis, as an idiopathic disease, is rare; it is generally connected with parturition, as we shall presently mention, but it may arise, in the unimpregnated uterus, from operations for polypus, &c.; also we have seen acute metritis and peritonitis caused by cancer of the uterus, perhaps through caustic or astringent applications; it is said to come as an aggravation of chronic catarrhal metritis, but we have never seen this. It is also said that metritis of the fundus causes the painful affection known as "irritable uterus." As a chronic disease it induces hardness of the organ with or without thickening, but this does not show very plainly in the fibrous-looking texture of the uterus.

Inflammation of the mucous surface, or *endometritis*, is believed to be the cause of some forms of dysmenorrhœa, particularly those where at every menstruation a discharge of membrane takes place, as you see in these bottles. It is still a question whether these arise as inflammatory exudations or by such an alteration of the mucous membrane as occurs in the formation of the decidua. They consist mostly of layers of pavement epithelium, and this has started the conjecture that they are formed merely in the cervix; thus this piece of membrane, when placed under the microscope, is found to consist of the most beautiful layers of flattened epithelium, and reminds one of the diphtheritic casts from the throat which are now so constantly being seen. Sometimes, however, the membrane has the exact shape of the uterine cavity, more frequently it is in several pieces, and when microscopically examined in thin sections it shows all the constituents of the uterine mucous membrane, loops of vessel running through it and glands being plentiful, while the stroma is a delicate tissue full of spindle and round cells, thus showing the characters of a true decidua. Many good authorities still believe that such casts as these never occur except through early abortion.

The post-mortem appearances of endometritis are seen in a softened bloated state of the whole organ from congestion, while in the interior the mucous membrane is red or purple, or sub-opaque, whitish, or thick, the glands enlarged either singly or in patches. More severe endometritis is found along with the pellicular and ulcerative inflammations of the vagina in fevers, &c.; you then may find the mucous membrane spotted over with follicular ulcers or ragged with more extensive ulcerations; sometimes the uterus is implicated in neighbouring abscesses as of the ovaries, and may be perforated by such an abscess, but this is rare. Sometimes portions of the mucous membrane, in which are, perhaps, several large glands, rise up as distinct elevations, or even project in a polypoid form, at last forming the mucous polypi of the uterine cavity which become much congested on their surface, and may give rise to great hæmorrhage through the thin dilated state of the vessels on their surface. Of these we shall speak presently.

Grave destructive ulceration (phagedænic) sometimes attacks the neck of the uterus, generally in old or weak subjects (as in this case, with similar disease of the mouth).

The inflammatory diseases of the *os uteri* are separate affections, and constitute, according to some obstetricians, a very large part of female disorders connected with vaginal discharges and various uterine disturbances; the lips of the uterus in these cases being said to be frequently ulcerated or abraded, or their follicles in a state resembling acne. These conditions do not constitute fatal disorders, and this is the reason, we suppose, why they are not more constantly met with after death, it being quite the exception to find ulcers in the uterus removed from the dead body; we do, however, meet with less degrees of disease where the *os* is congested or granular, and the epithelium even abraded; conditions which, owing to the turgidity and vascularity existing during life, may then present a much more striking appearance than when seen after death. Syphilitic ulceration is rarely so high up as the neck of the uterus, but it has been met with even within this. Such an occurrence is very rare, however.

Inflammation of the connective tissue about the uterus, *parametritis*, is not unfrequent. Cellulitic and peritoneal abscesses may be formed, and may burst into the vagina, bladder, or rectum.

The *os uteri* undergoes various changes from labour; it is seen to be puckered or fissured, or even absolutely closed, so that, as a preparation before us shows, the lips of the organ came quite away at a subsequent confinement. It also becomes narrowed by a chronic inflammatory induration of the lips or cervix, and thus arises one cause of painful menstruation, as you see in this specimen. A closure of the *os uteri*, generally the internal lip (*os internum*), may lead to a dilatation of the body of the uterus, as you may observe in some of our preparations. The dilatation arises from retained secretions; these may be purulent, constituting abscess within the uterus or *pyometra*, or they may be blood or menses, constituting *hæmatometra*. If the collection be of long standing it may undergo changes until only a brownish, perhaps fœtid watery fluid is left—*hydrometra*. Such dilatations are rarely of large extent, except in the case of retained menses or "hæmatometra," when the cavity may be widely dilated; but then the stricture is rarely at the os uteri. It is generally lower down the vagina, from congenital malformation, so that the vagina and the neck and body of the uterus are all distended alike, and the os uteri may be difficult to define.

Morbid Growths—*Myoma*.—We have already said, when speaking of the prostate, that new growths within it are of the same structure as that of the organ itself; and that this fact is analogous to what occurs in the uterus. This organ, as you know, consists of muscular fibre with some connective substance, vessels, &c., but in an unimpregnated state it is scarcely distinguishable by an unpractised eye from simple fibrous tissue. The tumours which grow within it are composed of the same elements, and when the organ is rapidly growing in size during pregnancy, so that new well-formed muscular fibres are becoming developed, then the tumour, if one be present, also increases, and its muscular nature becomes more apparent. The recognition of the essentially muscular nature of the tumours has obtained for them the name of muscular tumour or myoma, which has been substituted for the old name fibrous. The most common and convenient term for them is "uterine fibroid." The fibres are large, translucent, and solid, and appear flat like so many blades of grass. These uterine myomata were once called scirrhus, or, by Baillie, fleshy tubercle; they are exceedingly common, and we are almost daily meeting with them in persons of more advanced age. They are most frequent in the fundus of the organ, and when a section is made they are found imbedded in the tissue, forming round circumscribed masses. They are equally dense with or denser than the uterine structure, and the course of the fibres is more manifest; these running in a circular direction, and encompassing a centre from which the disease appears to have sprung. In a large tumour numbers of these concentric masses exist, and the fibres are seen coursing and curling in all directions, but more or less disposed to include certain definite spaces within them; sometimes several small tumours are seen when the uterus is cut through. Those that grow near the peritoneum project from the surface, and then slowly enlarge within the cavity of the abdomen; they are distinguished as the *subperitoneal*, and are generally the hardest and most prone to calcareous change; they exist for years, and there is scarcely a limit to the size they may reach, although rarely exceeding the size of the head, as you may observe by examining our numerous specimens. Very often a bunch of them grows from the surface, as you may see here, forming a mass of distinct tumours. At first these are continuous with the rest of the substance, but the connection becomes less as they enlarge, until they at last may become pedunculated, when they may be seen hanging by a slender cord to the fundus, and we believe they have even been found loose in the abdomen, though we have never seen this. They may, however, pass into the broad ligament and be found there apparently disconnected from the uterine tissue. The firmness of these tumours, or the hardness when cut with the knife, is sufficient to characterise them, and especially if there be any gritty or earthy matter within them, for this never occurs in softer or cancerous growths. Those which arise in the thickness of the uterine wall are called *intramural* or *interstitial*. They may reach a large size, and their growth may divert the course of the uterine cavity and give it a most curious shape. They may be associated with subperitoneal and submucous tumours of the same kind. They are generally not quite so firm as the subperitoneal nor so soft as the submucous tumours; sometimes, but rarely, a portion of such a tumour shows a great development of the blood-vessels, giving it the appearance of *cavernous* tissue. The myomata that form close to the cavity of the uterus constitute an important variety, as they tend to project into the cavity, and so to become "*polypi.*" They are rarely multiple, generally softer and more vascular than those in other situations with which they may be associated. They arise in connexion with the muscular wall, but as they grow they start away from it, and so may become purely *submucous* and press into the cavity, raising the mucous membrane before them, while the blood-vessels of the latter, and perhaps its glands, become much enlarged. We shall speak of their further history as polypi presently. Occasionally myomata ossify or, rather, calcify, when they become converted into round masses of bone. This change, as we have said, is more frequent in the subperitoneal tumours, but if it occur in a tumour which has grown inwards, forming a polypus (of which we shall presently speak), the uterus may be found filled with what is apparently a large calculus: a specimen of this you may see here. We have examined portions of this, and find the structure amorphous, having none of the characters of true bone; for this reason the mistake cannot now occur, which did some years ago, in supposing such a bony mass, which was found in a churchyard, to be an exostosis.

Cystic myoma of the uterus, or "*fibrocystic*" tumour, as it was formerly called, sometimes grows to a very large size, forming a great growth whose bulk, together with the fluctuation from fluid present in the cysts, may lead to its being mistaken for ovarian dropsy. When an operation was undertaken for ovariotomy under this belief, the uterus was sometimes removed, and this proving successful on several occasions the removal of such an uterus ranks among justifiable surgical operations. Under these circumstances the organ forms an enormous mass, in whose tissue are generally several tumours—one, however, being predominant in size. The tumour consists of the usual myomatous fibres, but instead of being homogeneous, its structure is opened up by spaces which contain a watery fluid, perhaps mucoid, and perhaps blood-stained; the spaces themselves are simply fissures or gaps in the tissue, and have no proper lining. They appear to be formed by a mere widening of the naturally microscopically small interstices in the texture. These cysts may prevail over the solid part of the growth, so that it is largely composed of fluid. They are generally by far most developed in the larger growths, while their earlier production may be well traced in the smaller. The cavity of such a great uterus is often enormously extended. If such a cavity were always permeable by the sound there would be no difficulty in the diagnosis of the disease, but, unfortunately for this purpose, the cavity is very apt to be bent out of its course or even occluded by the pressure and traction of the several growths, so that examination by the sound may appear fallaciously to confirm the diagnosis of ovarian tumour.

Polypus is the term used for the tumours which grow within the cavity of the womb in a more or less pendulous form. These polypi are mostly of two kinds: the one kind being those myomata which, as we have just described, commence within the uterine walls, and instead of growing outwards project inwards; they constitute the myomatous or fibroid *polypi;* while the others, which form the more common kind, grow from the mucous membrane or submucous tissue, and constitute the *mucous or cellular polypi.* Occasionally a sarcomatous tumour projects in the uterus as a malignant polypus.

In the first form the tumour is hard, though not so hard as those uterine fibroids which grow from the outside of the organ. When enlarged to a considerable size the more superficial parts of the tumour are altered by congestion, and, perhaps, exposure at last, so as to be soft and spongy with dilated vessels and œdema; or even to break down into partial cysts or abscesses, or else by simple incompleteness of the formation of the elements they are so soft as to suggest not inaptly the old comparison with boiled udder. These polypi always become more vascular than the external fibroid tumours, but the hæmorrhage which occurs from them during life is mostly from their mucous coverings; in some instances, however, it has been traced to an exposed vessel at the root of the tumour. This fibroid polypus, from commencing within the walls, is at first thoroughly incorporated with them, but in growing it tends to separate itself more from its attachments, and succeeds more or less in this according to its original nearness to the lining. As it grows it expands the uterus. A few of them may form more or less pendulous polypi, or even may be spontaneously ejected or slough away, but generally the continuity with the uterus is broad, and the growth retains its original seat, while the uterus becomes stretched over it until it sometimes reaches the size of the head; the tumour may now begin to slough on the side towards the os, and a part tend to escape, but operative measures being generally unattended with success, and in most cases quite impossible, death occurs from repeated hæmorrhage; or the attempts of the mass to escape may evert the uterus; we find after death the uterus distended to a globular form surrounding the tumour, which is attached to it and intimately blended with it by perhaps half its surface. Still, it appears that sometimes these tumours diminish greatly or even disappear. partly by sloughing, perhaps, but also through a tendency to simple degeneration, which they share with the fibre of the pregnant uterus, that they so much resemble.

Fibroid polypi are fortunately less common than those more manageable *mucous polypi*, growing by slender peduncles from the internal surface of the womb, which you so often see removed by the obstetric physicians, with which also we are so constantly meeting in the post-mortem room. You see them hanging from the internal surface of the uterus by a peduncle, and their most favourite seat is the *cervix uteri*, more frequently towards the uterine cavity, but some grow near the os externum, and more rarely they come from the fundus of the uterus; sometimes you find them from both together. As soon as they attain any size they pass into the cervix and protrude from the external os. Their attachment is generally quite slender, their form depends on the length they grow to; thus, at first they are nearly sessile and appear then as mere elevations of mucous membrane with one or more enlarged glands, when they are more properly called *mucous polypi*. They afterwards project and droop until they reach the shape of conical eardrops, or else are more globular, especially when they contain cysts, carrying then down some of the submucous tissue, so that their structure is *fibro-cellular*. Sometimes you may see several of them in the same case; thus, the other day we met with four in different stages of development. This kind of polypus differs essentially from the muscular fibroid in being an outgrowth of and from the mucous membrane and submucous tissue; they do not reach a large size. You will find their surface covered with columnar epithelium. The internal structure is composed, microscopically, of delicate but well-defined fibres, with caudate cells connected with them at intervals, and all embedded in a relatively large quantity of serous or slightly mucoid fluid. But besides these components of the general mass you find not unfrequently that a small polypus has one or more, perhaps a number of, transparent cysts or vesicles on its surface; from the fact of these being sometimes found in the cervix, it has been concluded that they might have been formed from Naboth's glands, which, when very large, as existing in a pregnant uterus, present a somewhat similar appearance. This is probably true, for intermediate stages of growth can often be found, but the cystic development is not limited to Naboth's glands, for you see cysts in polypi from the body of the uterus; in fact, this same uterus contains a cystic polypus within the cavity of the body, like that in the neck; and in this other specimen a similar cystic tumour, though small, is seen near the fundus. It may be that they are sometimes distinct formations, as in cysto-sarcoma of other organs, but it is probable that they originate in the glandular structures, as Dr Oldham considered; the intermediate stages we have several times seen, so that, we think, this is most frequently the case. Besides the varieties in the polypi, through the presence or absence of cysts, you meet with very different degrees of vascularity; they give rise to an extent of bleeding and distress very disproportionate to their size.

Fibrinous polypi.—Polypi of this kind have created much interest since the discovery of their hæmorrhagic origin by Velpeau and Kiwisch. They are composed of laminated fibrin, and are found in the uterus in cases of previous hæmorrhage, and it is believed by some never without an abortion; while others hold that menstrual hæmorrhage is sufficient to produce them. Some of the recorded cases support the latter belief, and yet there is no doubt that they are generally connected with abortion or delivery. Virchow says he never found them except under these circumstances. In appearance and in the manner of formation they closely resemble the so-called aneurisms within pulmonary vomicæ in phthisis. They are composed of fibrin laminated concentrically, to the exterior of which ordinary blood-clot may hang in greater or less quantity. In one interesting case we traced such a polypus to a varicose vein when there was no placental sore at all, but the whole mucous membrane healthy, and when besides this ruptured varix another similar uterine varix had ruptured into the peritoneal cavity, so causing death. Otherwise, the few cases we have seen have been after abortion or delivery.

Sarcoma.—It is certain that the internal organs may produce sarcoma, and we have now seen several examples of this disease in the uterus; here is one of them. The texture is composed of spindle cells, and is not so firm as that of simply fibrous tumours; it is softer and of a whiter colour; it does not, however, yield any milky juice like cancer when scraped, and in its mode of growth takes a place midway between a simple polypus and true malignant disease; for without destroying the whole tissue of the organ, like the latter, it springs from a large part of the surface of the uterus, and i attempts to descend has completely everted the organ; the edges of the growth are sloughing. It would have been quite impracticable in an example like this to have removed the tumour, and in cases where this has been done it has returned; in one instance, after the existence of the disease for some years, it proved fatal by secondary deposits in the lungs. In another case we met with recently the disease, which was, as usual, in the body of the uterus, had invaded a neighbouring coil of ileum. But sarcoma of the uterus is a rare disease; the growth is sometimes so hard as to be scarcely distinguishable from uterine fibroid, without the microscope to show its peculiar spindle cells. At other times it is soft, being of the medullary kind, composed of small spindle and round cells, with very little interstitial substance. We have seen cysts within a sarcomatous tumour in one case; also extensive hæmorrhage in another example, in which there were all gradations of hardness and softness from above downwards in the sarcomatous mass, which implicated nearly the whole body of the organ. In one, sarcoma coexisted in both ovaries.

Warty growths.—Both simple *fibrous papilloma* and *syphilitic condyloma* may be found upon the neck of the uterus. The relation of the larger warty growths of this simpler kind to the epitheliomatous cauliflower excrescence at this part is at least as uncertain as in other situations; you find the forms graduate into each other, and it is probable that in the individual instances a transition from the simpler to the more aggravated growth occurs; but it is very difficult to prove this.

Syphilitic gumma is said to have been occasionally found in the uterus, but it is extremely rare; we have never seen it.

Angioma.—Virchow describes a single case of small angioma of the uterus.

Carcinoma.—This is usually divided, as elsewhere, into epithelial, scirrhous, and medullary. Varieties do, indeed, exist, and yet the character of the disease which attacks the uterus is remarkably uniform; in the great majority of cases the malady being the same in its seat and general characters. The disease begins in the cervix, causing an induration of its lower part and of the os, and after a certain amount of infiltration a superficial softening takes place, and an open cancerous sore results; this gradually extends upwards and downwards, eating away the tissue of the body of the organ as well as the upper part of the vagina; in bad cases the disease penetrates to the rectum behind, or to the bladder in front, as you will see by many of our specimens. The patient dies at this time, or even before, from exhaustion, and on post-mortem examination we find, in most cases, that no other part is involved in the disease, the cancer having been altogether local. What, then, is its nature or degree of malignancy; is it like the more virulent medullary form, or the local epithelial cancer? This can scarcely be answered until the whole subject of cancer is more understood; the general opinion has been very conflicting, tending, perhaps, rather to group it with epithelioma, on account of its local nature, and from death resulting, as a rule, without any other structures being involved than those continuous with the original seat of disease. On the other hand, there is much reason to place it with medullary cancer, both because occasionally secondary deposits may be found in the lumbar glands, kidneys, liver, &c., and especially because the adventitious structure is composed merely of simple cells, and does not generally show those laminated capsules which are peculiar to epithelioma.

We speak, so far, under the impression that the constancy of the general character of uterine cancer produces; indeed, you must, after a short experience, be struck with the monotonous uniformity of these cases. Yet it is true that microscopic observation shows some variety in the structure of the morbid growth; nay already, without such help, we find some of them hard, so as to deserve the name scirrhous, while others are soft and strictly encephaloid, &c. The microscope shows, indeed, the usual difference in these two kinds of carcinoma, the former having a larger proportion of fibrous meshwork, while the latter has a vastly greater proportion of cells. On the other hand, some examples approach more to the characters of ordinary epithelial cancers, though we have never found any that quite resemble this growth as it occurs in the lip, œsophagus, &c.; thus the cells grow large and flat, in the form of squamous epithelium but multiform and bolder in outline with larger nuclei and more nucleoli, while the whole structure puts on a markedly glandular form, &c.; but, as we have said, the bird's-nest cells are either absent or small and doubtful. The distinction of epithelial from other forms of carcinoma is by no means clear and decided, and uterine cancer we have found to yield more doubtful and intermediate examples than any other kind we have examined.

Mr Arnott, after careful observation of twenty-two cases, came to the conclusion that epithelial cancer formed about three sevenths of them, and that fewer remote extensions to the ovaries, liver, lungs, &c., occurred in these cases; the rest were almost all medullary, and these infected remote parts more freely. In confirmation of this we have certainly found the uterine cancers which have implicated the ovaries, liver, cranium, &c., to be of a decided medullary kind, but we have not met with well-pronounced forms of ordinary epithelial cancer. The usual appearance shows a meshwork of fibres more or less slender, enclosing masses of cells which approach more to the cells that line mucous follicles than any other. The spaces too, are often of regular figure, so that the resemblance to a complex glandular tissue is frequent.

The kind of effect produced in the uterus and parts around varies a good deal. Sometimes the cancer perishes almost as rapidly as it grows, so that there is early a great excavation, the destruction of the tumour being so quick that you may find little more than a great hollow with scarcely a trace of cancer to be seen. The ureters may become implicated, and suppuration then reach the kidneys. We have seen the uterus thus eaten away until only a little piece of the fundus of the size of a shilling remained. The rectum and bladder are in such cases freely eroded. At other times the tumour tissue persists, and we then have seen it block up the neck of the uterus so as to obstruct the exit of secretions and so produce "pyometra" and purulent inflammation of the uterine sinuses ending in peritonitis. Sometimes the remainder of the uterus is thin and dilated. At yet other times the production of the cancer is so excessive that it forms large masses, which project into the vagina; these, however, are generally prevented from appearing on the post-mortem table by the care of the obstetric surgeon. In a remarkable case we have seen the cancer extend in the mucous membrane of the uterus so as to line its whole interior with a soft carcinomatous growth. We have known it produce irritation and suppuration of the iliac glands.

We have said that ordinary cancer of the uterus begins in the cervix, and kills the patient before the fundus of the organ is affected. We should mention, however, that cases are recorded where the disease has begun in the fundus. We have met with one such case associated with cancer of both ovaries, as it has been in other instances, as though it were secondary in derivation. In one case the body of the uterus was perforated by sloughing of the cancer, so that the peritoneum was reached; however, in such a case you must first make sure that the organ was not secondarily affected, for such has been the fact in all other instances of the kind we have seen. Thus, in a case we examined, a woman had died after the removal of a tumour from the abdominal walls, from a return of the disease within the body; and there we found the lumbar glands, ovaries, and body of uterus involved in one cancerous mass; in this instance, if we had not known the history, and seen the scar of the operation on the abdomen, it would probably have been thought that the uterus had been first affected; but the history of the case showed rather that the lumbar glands were the first internal parts involved.

Cauliflower excrescence.—This is not common, but you will often hear it spoken of as a peculiar form of disease of the uterus; it resembles, we believe, the papillary or warty growths on the penis which we have described, both in their general appearance and minute anatomy. It throws off a watery discharge from its surface, and is found on examination to consist of papillæ containing minute ramifications of arteries; we have already said that a warty character may belong to an innocent or malignant growth, and as regards the present case, although we have had no opportunity of making a thorough examination of this rare disease, we are told by obstetric physicians that it has a basis of epithelial cancer, and that it returns after removal. But these growths are so sure to be taken away by the obstetric surgeon that they scarcely ever appear at post-mortem examinations.

Tubercle.—It must be a question whether this ever occurs as a primary disease, for it is seldom recognised until after death, when it is found along with other tuberculous conditions as part of a general tuberculosis, and even in those where we have seen it suspected during life, the patient has evidently been suffering from phthisis, or tuberculous disease of the abdomen. The malady does not exist as tubercle within the muscular walls of the organ, but is almost entirely limited to the inner surface; thus, as you see here, the mucous membrane is converted more or less into this yellowish soft granular matter; on attempting to scrape it off it is found intimately blended with the tissue, and no distinct mucous membrane can be detected between the deposit and muscular walls, the membrane having been lost or destroyed in the morbid process. In specimens showing the earlier stages you find distinct tubercles in the mucous membrane scattered about; these break down and form yellow-based ulcers, which extend and multiply until the whole lining membrane of the body of the organ is destroyed, but you will find almost invariably that the disease does not affect the cervix, but stops at the os internum, a fact long ago observed by Dr Oldham. In cases of tubercle of the uterus you will, as a rule, find that tuberculous peritonitis is associated with it, and that the ovaries and Fallopian tubes have been especially involved; the latter have been much diseased and filled with the same scrofulous material, which would lead us to infer that the disease, beginning within the abdomen, has extended to the interior of these tubes, and then has crept down to the uterus; and so this organ is only affected by the spread of the peritoneal disease.

UTERUS AND PARTS AROUND AFTER PARTURITION

We shall hereafter speak of puerperal fever, but we may here bring before your notice the condition of uterus seen after labour. In an ordinary case you find the organ rather soft, and the interior presenting somewhat the appearance of an open wound; you may find much blood in it. Otherwise the surface is covered with a reddish-green fluid, and the spot where the placenta was attached is soft, raw, and pulpy. You must remember that the merely raw surface may be easily mistaken for a severe inflammatory patch when it is really ir natural condition. We have found venules open in the placental sore, and blood in the uterus six weeks after delivery. The tissue of the walls of the organ, too, is undergoing changes. You know that, while the uterus is growing so rapidly in pregnancy, new muscular fibres are formed in great numbers; after labour is completed a rapid degeneration goes on; and if you examine the structure beneath the microscope, you will find the fibres containing fatty granules, showing the degeneration which has already commenced preparatory to their absorption.

In women who die after labour various morbid conditions are found; in puerperal fever there is often no more local disease than an extreme softness of the uterus, so that the interior can be scraped up into a pulp. But in other cases of the kind you often find more or less grave inflammation of the uterus and its neighbourhood, and it is always a natural question whether, as these are accompanied by pyrexia, the name puerperal fever belongs to them as well as to the cases where the disease is fatal without, or with but trifling signs of local complication. As to this question we would remind you that in proportion as the fatal disorder is explicable by local injury and traumatic fever, in such proportion is it of a different kind from fever proper, which begins in a general blood change, and hence we agree with those who draw the strongest possible distinction between the local traumatic inflammatory *metritis*, *cellulitis*, &c., on the one hand, and the properly *febrile disorder*, puerperal fever, on the other. You have heard Dr Hicks's reasoning from his experience, which goes to show that much of the puerperal fever arises from exposure to scarlatinal, diphtheritic, and other poisons; there can be no doubt of its intensely contagious nature.

In many fatal puerperal cases, however, you find decided *metritis*, a turbid fluid or even pus being in the uterine wall. It is rare to meet with this throughout the whole of the organ, but it is very common at the seat of the attachment of the placenta or about the injured cervix. Inflammatory products very rarely take the form of abscess in the uterine wall, but we may find small lines or strings of small cysts of pus in the lymphatics or purulent thrombosis of the veins of the organ. The peritoneum over the uterus may be inflamed either at the same time as the interior, or, in some cases, without any evidence of the interior partaking. In these cases the Fallopian tubes are often inflamed within so as to contain pus or muco-pus, and it is a question whether the peritonitis is produced by extension of inflammation from the interior of the uterus along the tubes. We have seen *perimetritis*, as the peritoneal inflammation is called, when there was no sign of *endometritis*, and it is possible that the inflammation may extend from the peritoneum into the tubes.

In other cases, where the interior of the uterus is acutely inflamed, the inflammation lights up outside the muscular wall in the subserous connective tissue about the uterus and in the broad ligaments, which becomes soaked with turbid serum, or suppuration may arise there and extend into the pelvic cellular tissue, constituting "pelvic cellulitis." Abscesses so formed, usually extend upward so as to be opened in the iliac or pubic region, and may rise very high. They more rarely burst into the vagina or rectum. We have seen several deaths by exhaustion when the latter has been the course, whereas recovery is the rule in cases which discharge externally. Such abscesses may find their way into the bladder or perinæum, but they *almost never* burst into the peritoneal cavity. They may be evidently started by direct injury to the contents of the pelvis during delivery; thus they may arise by the suppuration about large blood-clots caused by rupture of vessels. They obtain various names from special writers. The most convenient are those compounded of the name of the organ they are near with the prefix *peri*, such as *peri-cystitis*, *peri-proctitis*, *peri-vaginitis*, *peri-metritis*, but Virchow has used the latter term for the peritoneal inflammation of the uterus, employing the term para-metritis for this subserous form. This is a little unfortunate, as it taxes one's memory for a disharmonious use of the term.

Puerperal Thrombosis.—Three chief conditions favour the formation of thrombus in and about the uterus at parturition; firstly, there is the pressure of it on the iliac veins, &c., tending to induce stasis and coagulation about the valves and circumflex veins within the femoral vein; secondly, there is the greatly dilated state of the veins of the uterine plexuses, inducing stasis; and thirdly, there is the coagulation in the uterine sinuses at the placental sore. Either of these three conditions may produce *ante-mortem* coagulation or thrombus, and the three kinds of such clots are distinguished by Virchow as the *compression-*, *dilatation-*, and *placental*-thrombosis respectively. Two dangers of different kinds attend them; *first*, they become spongy dead masses, which absorb foul materials either from the sore or from the blood, and when they disintegrate yield them back in a more pernicious form, producing ichorrhæmia; and *second*, they may be dislodged *en masse* especially from the femoral veins, and be impelled into the pulmonary artery, producing late embolism after delivery. The earlier embolism, described by Dr Playfair and others as thrombosis of the pulmonary artery, we believe to arise as ante-mortem clots in the depression and faintness of parturition, which form in the apices of the ventricles or in the auricular appendices, and are at once dislodged and thrown into the pulmonary artery.

Rupture of the Uterus.—This sometimes occurs during the expulsion of the child, and a large rent results, as you will see by the numerous specimens in our museum. The rent, you will observe, passes through the os, and extends some way up the walls of the organ, and in most cases runs up obliquely on one side, and sometimes downwards into the vagina. It is generally several inches long, and causes death by hæmorrhage; but sometimes it is less complete, and the bleeding may show itself in the form of a soft tumour in the neighbourhood of the bladder or outer abdominal wall. It may occur in cases of contracted pelvis, or where the uterus is ill-placed for delivery; when labour is unassisted, or when instruments have been clumsily used; or it may be the result of other violence, less well-intentioned; but in most instances, at least this has been the history of our recent cases, there has been no mechanical difficulty, and the subjects have been mothers of several children; and therefore the cause appears due rather to some defect in the tissue of the organ. We do not know, however, that this has been positively proved. There is some difficulty in deciding the question, for, as we have already said, the uterus always shows a degeneration of the muscular tissue at the completion of gestation, and then, even with the microscope, it is hard to say whether this is in excess, or whether it has commenced prematurely in any individual case. We have examined several examples, and in none was there any very marked disease of the tissue. In this strait some adopt a belief that the uterine walls are thin at the part that gives way. Dr Barnes has pointed out the contraction of the uterus on a dead child as an important cause of rupture.

We have never seen a case of rupture from disease, although we have heard of such an accident from tuberculous affection during the course of gestation, also of a case where there was cancer of the neck of the organ.

The wound of *Cæsarian section* generally gapes widely on the peritoneal aspect.

Hydatids.—*Echinococci* have been found in or have escaped from the uterus in several instances, as in the example recorded by Dr Hicks.

FALLOPIAN TUBES

Malformation.—Both tubes may be absent along with the uterus, or this may happen on one side only, or the tube be but ill developed, the uterus being deficient on the same side. The fenestrated end varies in its complexity, sometimes being little developed, at other times large with two or three openings.

Most of the diseases of these parts are secondary to morbid conditions of the ovary, or inflammatory processes in the pelvis, or adventitious growths in the abdomen, &c. Thus, they may be enormously lengthened by ovarian cysts; cancer in the lower part of the abdomen involves them in the disease, and in tuberculous affection of the peritoneum these parts rarely escape; also pelvic cellulitis, arising from whatever causes, generally implicates them.

Inflammation.—In cases of pelvic cellulitis, or suppuration about the ovaries, the free extremities of the tubes often become closed and adherent to parts around; and, as the same process implicates their interior, the result is that the uterine ends also close up, so that thus shut up at each extremity the tube becomes enormously distended with purulent matter, as you see in this instance, or else with serum or sero-purulent fluid. The results of a more chronic inflammation, as seen in mere adhesions, are constantly met with; the fimbriated extremity of the duct is united to the ovary, and all these parts are much contracted or puckered by the ancient local peritonitis. It was observed by Morgagni that this condition was very commonly found in prostitutes, and was suggestive of a cause of sterility, and our own experience quite confirms the statement. It appears probable that the cause of this condition is usually gonorrhœa, which extends along the Fallopian tubes to reach the neighbourhood of the ovaries, just as it sometimes extends along the spermatic ducts to reach the testes. In one of our cases it coexisted with chancre in the vagina.

Tuberculous Disease.—When speaking of tubercle of the uterus we noticed the way in which the Fallopian tubes are involved by extension of tuberculous peritonitis; the tube then becomes distended with a thick, curdy, or scrofulous matter. In early stages the wall can be seen to be sprinkled with miliary ulcers from softening of tubercles in the mucous membrane. This we have seen in two cases. The disease is most severe at the peritoneal end of the tube, and its extension is downwards to the uterus. We have never seen it except in association with tubercle of the peritoneum; but it may be that the extension is sometimes from the tubes as the primary seat of attack to the peritoneum secondarily. When there is much fluid in the peritoneal cavity the thick fimbriated ends of the tubes may stand rigidly up in it, looking like sea anemones.

Morbid Growths.—*Fibrous tumours* rarely form in the tubes, also *Myomata* in them are rare and small.

Cancer.—This may arise by contiguity from the tubes being involved in cancer of the abdomen; you will never find them affected by an extension of the disease upwards from the cervix.

Cysts may be found hanging from the Fallopian tubes and broad ligaments. These are generally small, not exceeding the size of a cherry or walnut; they are usually regarded as fœtal remains. Thus kind of cyst is single, and is frequently present close to the fimbri edge of the tube; this is traced to the so-called "Müller's thr on the Wolffian body, representing its dilated end, the rest of it used for the Fallopian tube itself. Other cysts may be multiple placed on the broad ligament at some distance from the fimbriated that is, in the seat of the "organ of Rosenmüller" or remains of Wolffian body. Accordingly these cysts are viewed as dilatatio those relics, but a few other cysts yet, sometimes larger, are foun a distance from the organ of Rosenmüller, or even, it is said, wi the broad ligament, and these cannot be explained except as products.

Cholesteatoma.—We have on three occasions met with these s cystic tumours on the broad ligament filled with pearly scale cholesterine and detritus of epithelium, &c. They are of no cli importance.

OVARIES

Malformation.—The ovaries may be absent or ill-developed. failure of development may affect one only, and may coexist equal imperfection of the rest of the internal genitalia of that The ovaries may too, as we have already said, pass into the groi labia. They may even find their way into the femoral ring.

Atrophy.—We sometimes have met with small perfectly sm ovaries not one third their natural size, in sterile women. These s ovaries were surrounded by relatively large radiating veins.

No other organs, perhaps, suffer so much defacement in the ordin performance of their functions as do the ovaries; the enlargem and bursting of the Graafian follicles, and the frequent hæmorrh into their interior, is designedly a rough process, and thus it pla contains one cause of inflammation; for, however normal laceration bleeding may be, as in parturition, the violence done is neverthe equally violence, and in presence of other irritations it plays its towards setting up disease. Hence the thickened capsule and puckered fibrous cirrhosed remainder of the ovary generally foun middle age. It is probable enough that such subinflammatory fib states occurring in earlier life through greater irritability may b cause of sterility; this we cannot say, but it is a subject well wor of investigation.

Extravasation of Blood and Corpora Lutea.—It is very common find the Graafian vesicles filled with blood; such bleeding is suppo to be connected with a discharge of ova at the menstrual periods.

we meet it too early for this, as at seven years of age; we sometimes find clots, even, say, of the size of a cherry, associated with a sanguineous fluid in the uterus, in those who have died of congestive disease when not menstruating, as of morbus cordis, or of fevers. When such hæmorrhage as this occurs, the inner of the two walls of the Graafian vesicle thickens a little and grows yellowish, and after the blood has existed in the organ for some time, the usual changes occur in it; it shrinks and becomes brownish and yellowish, so becoming like the *corpora lutea* found after a true conception; hence such patches are called *false corpora lutea*.

The true corpus luteum is found when an ovum has escaped, and its development is proceeding in the womb; a characteristic change then goes on in the ovarium itself: one of the Graafian follicles (or two, if there be twins, for the number corresponds in all animals with the number of fœtuses) is found to be greatly enlarged. When at its full size it is a globular mass of the size of a marble; this, when cut through, is found to consist of a softish yellow matter surrounding a central part of a pinkish colour, the diameter of the latter being about equal to the breadth of the former. What has happened to the follicle is this. The inner of its two membranes has been swollen so as to form a soft, yellowish, vascular substance, which, by the increase of its surface, had to fall into folds to accommodate itself to the follicle. At the same time the outer membrane and ovary around grew more vascular. The thick folded inner membrane reduced the cavity within to a small, irregular cleft, of a whitish colour, and thus what you see on section when the body is fully developed is a yellow substance surrounding a white stellate or zigzag line. On the surface of the ovary, also, a cicatrix is seen. If you examine the section microscopically you will find, as you see in this sketch, a slightly fibrillated material, made up chiefly of fatty, cellular, and fibrillar matter, with a few capillaries; the fat gives it the yellow colour, which is increased by some blood in an altered condition. The body increases until it reaches its full size in the third or fourth month of the fœtus; it then becomes smaller, at the time of natural labour it is much reduced, and in a few months has altogether disappeared. Thus its importance would be only as one amongst the evidences of abortion in the early months of pregnancy. It is distinguished from the false corpus luteum of menstruation by the greater development of the inner folded membrane which causes it to be larger and to project more boldly on the surface.

We may here mention that sometimes blood has collected within an ovary, and so distended it that a *hæmatocele* has been formed. Rokitansky has shown that this is probably caused by a twisting of the pedicle of an already enlarged cystic ovary which strangulates it, and so leads to the bleeding. We have never met with a case, but some examples are recorded. The bursting of such ovarian blood-cysts proves fatal, or the bleeding of a strangulated ovary causes sometimes retro-uterine hæmatocele.

Inflammation and Abscess.—Ovaritis may be either a primary or a secondary affection. In many of those cases we meet with in the wards, of local peritonitis in the lower part of the abdomen, connected with some strain or chill during a menstrual period, it is known that a primary inflammation of this organ has occurred. When the ovary is inflamed the tissue becomes soft and reddish. The Graafian follicles have blood or other inflammatory effusions in them and pus may be seen in patches or lines, or if the process be less diffuse and grave an abscess may arise. The exposed state of the ovary within the peritoneum, so that the latter soon takes on the inflammatory action, renders such acute ovarian inflammation very dangerous, and thus it is that it becomes a rather frequent cause of acute peritonitis in young women. In these cases the disease is generally limited to one ovary, but if the patient survive and abscess form, the parts get so confounded by the chronic inflammation that it is scarcely possible to trace the disorder to its seat of origin. Also, in connection with parturition and pelvic cellulitis, the ovary may be involved in the suppurating inflammation, if it be not sometimes, indeed, the chief seat of the mischief; in some of those lingering cases of suppuration in the pelvis and about the genital organs connected with parturition, it has been pretty certain that the ovary has been the starting-point of the disease, although, when a number of parts become involved in one extended suppuration, it is difficult to unravel the mass to bring this to the proof.

Both ovaries, nay, indeed, all the internal genitalia, are generally found affected by the time when these puerperal cases come under post-mortem inspection. We have met occasionally with pyæmic abscess of the ovary, and an abscess in phthisis, but no tubercle. We have mentioned to you that the Fallopian tubes are often bound to the ovaries by false membranes; this is generally in prostitutes, and is traceable probably to gonorrhœa.

We often find the Graafian follicles very thick and large, like boiled pea-skins. The exact consequences of this apparent chronic inflammation of the Graafian follicles we do not know; it occurs equally in multiparous or sterile females, and when the ovary is unadherent we have often noticed the follicles not to be ruptured. The suggestion of Rindfleisch that their thickness enables them to give counterpressure while the follicles dilate to cysts is interesting.

The ovary is very rarely the seat of syphilitic gummatous change; we have never seen it, but the adhesion of the ovaries to parts around is very common in cases of syphilis, perhaps through gonorrhœa intercurrent.

Morbid Growths.—*Cysts.*—We have already told you, when speaking of the kidney and testis, that much difference of opinion exists as to the origin of cysts; this difference of opinion reaches its most distinct opposition in reference to the ovary: the question being whether they be mere expansions of the original Graafian vesicles of the organ, or whether they be new formations. It is probable they arise in both of these ways. In very many cases there can be no doubt that the latter must be the true interpretation, for you find them growing in immense numbers, and forming in the walls of the parent cysts, where there is no trace of proper ovarian tissue. On the other hand, we are constantly meeting with ovaria which, when opened, show Graafian vesicles dilated with blood, or watery fluid, and which at once suggest an origin for cystic disease; and, indeed, the ovum itself has been found in cysts of the size of a bean. We have ourselves found an ovum in a cyst half an inch in diameter. Here, no doubt, is the explanation of the frequency of ovarian cysts. The specimens we show you exhibit these vesicles in various degrees of enlargement.

The distinction of ovarian cysts into the simple and compound, or unilocular and multilocular, as it is sometimes expressed, is a very old one and has certain practical advantages; yet we have not found it capable of any more than a rough application. You so frequently meet with ovarian cysts, which are to all practical intents simple, but yet have one or more cysts at some part, or at any rate show septa or other marks of having formerly been compounded of cysts that have ruptured into each other. Indeed, this is so frequent that it is doubtful whether any of the larger cysts are ever originally or quite single. It is certain that neighbouring cysts as they grow burst into each other by their mutual pressure and thus coalesce into one great cavity. The simple ovarian cysts are known as *hydrops folliculorum* or distension of Graafian vesicles with watery fluid. This is a comparatively unimportant condition, as the cysts never reach a large size; and, although it may be suspected, there is no evidence that these simple dropsical follicles ever become ovarian cystic tumours; when yet small, ova may be found in them.

Ovarian dropsy.—First, the proliferous form. You find a very large cystic tumour, generally unadherent, and with one or two or a few cysts predominating greatly in size, while the rest are much smaller, and at some part or parts there is usually a portion of more solid-looking tissue; seen on closer examination still to be composed of small cysts and representing the remains of the ovary. The capsule is thick, whitish, and fibrous, the interior of the greater cysts pale, and their contents glairy or more tenacious, often turbid and more or less deeply discoloured. The inner surface of the large cysts is softish looking and bears more or fewer knobs or excrescences of some form standing up in relief. The walls of the smaller cysts are more vascular, sometimes they have a honeycomb-like or cribriform appearance from fringes of membrane coursing about on them with variable divisions and reunions, while the more solid-looking parts and the section of the solid-looking excrescences look, at first glance, not unlike cancer, being pink, creamy, and vascular.

The appearances under the microscope differ according to the part chosen. The wall of a large cyst is fibrous, generally laminated, the inner part more delicate and areolar with vessels visible in it; the lining covered with epithelial cells, generally cylindrical; these are easily removed. The same cylindrical epithelial cells, perhaps ciliated, generally much defaced, float in the mucous fluid; but pus or blood may be present. If you take the wall of a smaller cyst it shows signs of greater activity, and is more full of vessels and the epithelium more regular. But the most striking characters of this kind of cyst are seen in the formation of bud-like processes within the cysts; they are best brought to view in the most solid-looking parts, and especially within the larger or smaller excrescences on the walls of the principal cysts. These microscopic bud-like knobs grow out and branch, uniting with each other, forming arches, or they extend across the cavity they are in, and thus spaces are enclosed which, in section at least, look very like mucous follicles, and they are lined with the cylindrical epithelium, which usually everywhere clothes the buds. The buds have vessels in them; they may branch and branch until they no longer are clothed with a distinct epithelium, but the twigs confuse each other and the cells are massed together like those of cancer. This we have seen in parts of a tumour where other parts showed the usual bud, and in this case there were metastatic formations in the liver, thus showing that this form of tumour, when very vivid and rank in its growth, will prove malignant like any other. There is some ambiguity in the interpretation of the appearances, some thinking the small enclosed cyst-like spaces are cysts about to widen, and others that they are rather, as we have just described them, compartments recently enclosed by uniting buds; both views are probably true, so that after the cyst is closed in, as it were, by new growth, it afterwards may dilate and itself bear new buds. This kind of cyst is by some believed to grow from Graafian vesicles, intermediate stages having been suggested by finding the other ovary showing a smaller cyst; in one such instance Rindfleisch found an ovum in a little cyst so related to one of these tumours.

The second kind, which are comparatively simple cysts, are essentially distinct from the kind just described. The coarse appearance, nevertheless, is very similar, at least superficially. You find a large cystic tumour more often adherent than the first form, its capsule thick and fibrous, almost round if there be one prevailing cyst; otherwise presenting as many convexities as there are large cysts, generally having at one or more parts a more solid mass in which the origin of the cystic process may be studied. In this case generally the small cysts in the more consistent part are more clearly conspicuous to the naked eye than those of the first kind, and are seen as definite spaces with watery contents. The microscope shows the origin of the cysts as simple gaps in the structure of the ovary, appearing at first as dilated fissures or interspaces in the texture. Some think these may be due to changes in very young ovisacs, before yet they are Graafian vesicles, but there is no proof of this. What you see is a group of fissures occupied by fluid, the fissures, widening, communicate with each other, reducing their partial separation to mere films crossing the spaces, which now assume a round figure with tolerably defined walls and contents of thin mucous matter, more or less limpid, generally more so than the contents of the first kind of cyst. There are no buds into the interior of these, and the formation of new ones can be traced in its several stages in the laminæ of tissue between the large cysts where there are not any Graafian vesicles.

We have mentioned that the coarse appearance of these two kinds of cysts is not very different; their contents are more or less glutinous; once we found them so tough that they required to be cut out of the cyst; they may be charged with blood in various stages of change, or, occasionally, with pus. The origin of the colloid substance is an interesting question; it may be secreted as a mucus from the surface clothed with cylinder epithelium. In a case of congenital sacral tumour with cysts lined with such epithelium, the contents were mucus. It is thought by others that the mucous matter is a result of a kind of digestion of the matters supplied by the neighbouring blood and tissues of the ovary.

Cases are recorded where ovarian cysts have spontaneously ruptured into the peritoneal cavity, and the patient has recovered. Dr Bristowe has shown that such rupture is not very uncommon.

We have met with a third form of ovarian cystic tumour in which were a vast number of cysts of all sizes embedded in a tissue of lymphomatous structure; the cysts could be seen to be first formed, by the appearance of a minute sphere of cells, in the tissue surrounding a portion of the structure, which enclosed portion afterwards perished, so that the sphere of cells became the inner surface of a young cyst, which then grew to a large size in two or three instances. The disease was symmetrical.

Excepting in a few cases of true cancer, to be mentioned presently, cystic ovarian disease is a local affection, there being no other disease in the body accompanying it; death is caused by exhaustion after repeated tappings, suppuration of the sac, extravasation of blood into it, &c. The tumour may be quite free from adhesions and attached merely by its peduncle; it very often happens, however, that it has formed adhesions to other parts, especially the second form; these adhesions are more frequent in connection with the anterior parietes of the abdomen, if paracentesis has been frequently adopted. The cyst may, however, be attached to the intestines lying behind it, and very frequently the colon is fixed to its upper part by the omentum, which closely covers it. It may thus produce intestinal strangulation. Tubercular ulcer in the intestine may open it. Sometimes also the ureters are found adherent to the cyst, and if not, they are very frequently dilated by the pressure exerted upon them, the dilatation extending to the pelvis of the kidney; the uterus too may have become attached, and if so, being forcibly pulled upwards, is much lengthened, as we have already shown you; the other ovary is generally healthy, and that it is really so is proved by those cases where, after the extirpation of the diseased organ, the patient has borne children, both male and female; but cases are recorded of an appearance of a second tumour after ovariotomy, requiring a second operation, which, in one instance, was successfully performed. We have specimens on our shelves showing instances where the ovarian cyst has formed communication with the colon, bladder, &c.

Fibroma.—Occasionally, though rarely, the ovary may be converted into a very hard solid fibrous tumour; this may contain cysts closely resembling those in cystic myoma of the uterus.

Sarcoma—Cysto-sarcoma.—More frequently these solid tumours are composed of a softer fibrous tissue and are vascular, resembling the uterine polypus. The one which is here is of the kind called fibroplastic, composed of spindle cells, and exactly resembling the recurrent fibroid tumours of external parts which are removed by the surgeons; in this one, which has a similar structure, a large cyst is formed, showing the general tendency to cystic formation in these ovarian tumours.

Carcinoma is not common as a primary disease; we have never found it reach a great size without cancer being present in other parts of the abdomen. We have often seen the peritoneum partake of the disease, just as it lights up into inflammation in ovaritis, so that cancerous ascites may clinically cover the signs of the ovarian disease that began the mischief. When ovarian cancer exists as a primary disease it forms, generally, symmetrical masses of opaque whitish nodulated growth, with veins ramifying on their surfaces. The size is rarely large, and the surface is not smooth and shining, like that of ovarian cysts, but more dull and uneven. When cysts form in it, as is not unfrequent, it resembles the cysto-sarcoma, only instead of being composed of spindle-cells, the texture contains nests of rapidly growing epithelial cells of various forms compacted together, with large nuclei and many bright nucleoli, and lodged in meshes of an alveolar tissue. The disease is then eminently malignant, and is propagated to other parts. In several cases of this kind which have occurred here, there was generally no suspicion of the disease being malignant except by its rapidly fatal termination within a few months; and after death the tumour was multilocular, and contained solid growths as in ordinary ovarian disease; but the liver, lungs, and other parts were filled with cancer. Such a case must be called *cysto-carcinoma.*

Carcinoma of the ovaries is generally secondary to cancer of the uterus or of other parts; it is usually double. On the other hand, we have seen the ovaries resist a peritoneal cancer extending all around them.

Piliferous Cysts.—The cysts known by this name are characterised by containing hair, as well as generally fatty matter, with sometimes teeth and amorphous pieces of bone. They have no connection with the class of diseases of which we have been speaking, and yet we have met with them sometimes, as it were, casually associated with simple cysts and with malignant disease. In a very remarkable case recorded by Mr Hulke there were numerous secondary dermoid cysts about the liver; the primary disease being cancer associated with dermoid cyst about the ovary; also in a case by the late Mr Moore there were many small dermoid cysts in the omentum, &c., near a large one. So that it appears they may prove infectious, at least like cartilage, when associated with malignant growths. These cysts are generally met with accidentally in persons who have died of other disorders; although, indeed, in a case that occurred lately one of them was the cause of death by rupture and subsequent peritonitis; they are not generally larger than the distended bladder, if so large. The most frequent contents are white fatty matter and hair. The former is no doubt secreted by the sebaceous follicles found in the lining membrane of the cyst, and is fluid while the patient is alive, but becomes solid as the body cools;[1] in this bottle you will see the fat in the form of a number of perfectly round balls. This fat entirely dissolves in ether, leaving no membranous residue. The hair is in bundles, and is generally, we believe, of a light colour; when examined it is found to be pointed at both ends, but this probably is an acquired condition, for no doubt it has been formed on the surface of the cyst, and thrown off. In the specimen which occurred here lately, there is a good cuticular lining, and from it a number of hairs are growing; a section of the skin shows well-formed hairs in their follicles, and a very rich supply of sebaceous glands, which were no doubt constantly pouring out their fat. In this specimen there were also three teeth imbedded in a piece of bone, which Mr Salter thinks have been there many years, although they are the primary or milk teeth, and, what is remarkable, he thinks the teeth contain in their pulp true nerve substance. In the Warren Anatomical Museum are two bones from such a cyst, with seventeen and thirteen teeth in them respectively.

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[1] This has often been surmised, but never, as we believe, proved. Since this was written, a case has occurred where the contents of the cyst, when opened after death, were fluid, but soon solidified into a mass like tallow.

We will not take up your time with the various opinions given as to the origin of these remarkable tumours, containing within them the parts necessary for the construction of a fœtus, but we may mention some theories raised to account for them. First, one founded on Owen's view of "*partheno-genesis*," in allusion to those kinds of insects who, from one impregnation, continue to bring forth successive generations; suggesting that in the human body, in an exceptional case, the fœtus when born of its mother might contain within it the germ of another fœtus. A second theory is that which assumes an *included ovum;* where, after the impregnation of the ova, one shall envelope the other wholly or partly, and thus one shall be found projecting from the other (as we shall presently show you). In these cases, however, the included ovum is either a heterogeneous mass, or shows merely a resemblance to a fœtus, and may be met with in any part of the body. It has also been thought that they spring up in the ovary as an independent act on its part as a *generative* organ, but dermoid cysts are not peculiar to the ovary; they occur in other parts, even the brain and its membranes, and, as we have seen, may be repeated by malignant infection, so that it appears they spring up in the ovary in the same way as elsewhere, and as obscurely as other tumours.

MAMMA

Hypertrophy.—The natural increase of the gland in lactation is one of the most remarkable examples of hypertrophy which could be instanced. Sometimes the breast grows to such an enormous size that its removal is required, and this without the formation of a distinct tumour in it, but as an uniform increase of the whole organ. This is very rare; in one case of the kind, the organ, when excised, weighed many pounds.

Atrophy.—In the decline of life the mammary gland atrophies, and in some old women, from a loss of fat and shrinking of the secreting structure, the remaining fibrous tissue has formed a hard lump, which might be mistaken for true scirrhus; at least, we have been questioned as to its being of such a nature more than once in the post-mortem room.

Inflammation and Abscess.—Superficial excoriations and fissures about the nipples form a very painful affection during lactation; sometimes they lead to erythematous inflammation. The skin about the nipples is liable to molluscum contagiosum; also to syphilitic ulceration and condyloma. The deeper inflammations you frequently meet with during lactation, and more rarely as idiopathic affections, or resulting from injury.

The inflammation may be either within the mammary gland or in the tissue around or beneath it. The tendency is to form abscesses which may be localised in some part of the gland or more diffused around it externally. They rarely excite pleurisy. They are generally opened by the surgeon; sometimes they burst in two or three places, and lead to a formation of fistulous canals. Some of these abscesses become chronic, and have been known to dry up, leaving calcareous relics.

Morbid Growths.—The female breast is frequently affected with tumours, and these are often complex, and differ much from each other in several distinct ways. Thus, they differ in some dilating the ducts into cysts, while others do not do so; and, again, they differ in being composed of different materials. The cystic dilatations are very striking to the naked eye, especially when pedunculated masses of the tumour of various forms grow into these cysts; but you find cystic tumours with very much the same appearance, composed of textures essentially different from each other; thus you may get cystic adenoma, fibroma, sarcoma, or myxoma, looking so much alike to the naked eye, that the microscope is required to distinguish them; and, again, you may find adenoma, fibroma, myxoma, &c., free from cysts, but essentially of the same nature as if these had been present.

A few years ago, when Mr Birkett proved that many of the intracystic growths are composed of a structure identical with that of the mamma, *i.e.* of "adenocele," the interest of this discovery caused it to prevail in the consideration of all intra-cystic growths; and as most of these growths contain some glandular elements, such elements were regarded as the predominant feature in the tumour, and hence it came about that these intra-cystic tumours were all called adenocele more or less confusedly, and the old term for them, "cysto-sarcoma," fell into disrepute. Mr Birkett's own plan of teaching was then to show to his class how the various forms of these tumours resemble the artificial dissections of the mammary gland, according as more or less of this uniting fibre tissue is taken away. Thus, a section of a female breast shows merely an uniform fibrous surface; but if by dissection some of the areolar tissue be removed, it is found to be separable into lobes; these lobes, again, by further dissection, may be separated into lobules; and these again, by a yet further removal of connective tissue, ir primitive acini or cæcal terminations of the ducts. Corresp all these degrees of dissection, so may the new tissues be developed in the adenoceles: in one case the gland tissue is at once seen by a magnifying glass, minute lobules being connected by ducts; in another these are clustered to form larger lobules, which are distinctly seen by the naked eye when the tumour is opened; and in another case the connective tissue so preponderates that the whole is conglomerated into one uniform mass. All these varieties you will see on our shelves, some of which, growing many years, reached several pounds in weight, and have very uniform solid structure, while others display the lobules of various sizes. The preparations were chiefly made by Mr Birkett's admirable dissections, and at the time when he taught these views they were in accordance with all that was known of the connective tissues spoken of as interstitial between the glandular acini.

But since that time the microscope has shown that the material in which the adenoid elements in the growths are embedded is itself of too great importance and too various to be ignored. So that while Mr Birkett's teaching as to the existence of glandular elements remains as true as ever, it is important to know that along with these elements others of a fibrous, sarcomatous, or myxomatous kind are apt to be present, and may greatly prevail and determine the character of the whole growth, so that the interstitial material comes to have a new consequence, and the question arises often, when the gland substance is in smaller amount, whether this may not be altogether old and represent included portions of the original gland. All these conditions have been carefully studied by Mr Birkett; you will find them fully described in his work on the breast.

The recognition of these connective elements of intra-cystic growths is carried to an exclusive extreme by some recent writers, as Rindfleisch. He teaches that the tumours with cysts into which growths project are of a totally different class from adenoid. So that in his main division, viz., into mammary tumours from epithelial growth, and mammary tumours from growth of connective tissue, he puts adenoid tumours in the first and cysto-sarcoma in the second, thus separating them entirely. We think that this view is wrong in its exclusiveness, for, as in this preparation, you do find typical glandular tissue forming growths into cysts, and this in a large number of cases and to a large extent. This fact he appears to ignore altogether. On the other hand, it is necessary to admit that the elements of these growths, which give them their great importance, are often the interstitial connective substances, and these may exist in such intra-cystic growths without any glandular substance at all, and they are various in their kind.

Another point much confused at present by histological authorities is the relation of the old adenocele or chronic mammary tumour, now called adenoma, to carcinoma. We will endeavour to show this confusion, for it must be avoided rather than cleared up by the student. You know that the definition of carcinoma is not very different from that of adenoma; indeed, it is scarcely possible to draw the distinction without going into details about very minute points, especially the characters of the cells, which are large and have large nuclei in carcinoma. Formerly these cells would have been thought to make a very sufficient criterion, but Virchow repudiated the "cancer cell," and very ably directed attention to the plan of histological composition of tumours as promising a more sure means whereby their nature could be determined, and since then the cancer cell has fallen into disrepute. But although there is no doubt that a cancerous action does belong to tumours, such as lymphoma and some sarcomas, which have no "cancer cells," yet in this particular question between adenoma and carcinoma it is very important to note that the cells of the latter are very different from those of the former, so that *as between these two kinds of tumours the cells do form important distinctions*, however comparatively unimportant they may be elsewhere; indeed, it is difficult to draw any histological distinction between these kinds without putting the cancer cells in a prominent place in it. The structure of the gland, as you know, when seen in section, shows its acini cut across, so that you see spaces lined with epithelial cells, the spaces themselves having proper walls of fibroid stroma, which form a meshwork in the section, defining the outlines of the spaces containing the epithelium; in whose walls a good observer will detect capillaries and lymphatic channels. Now, the structure of a cancer would in description correspond very much with all this; you have epithelioid cells in the interior of spaces which are separated from each other by a meshwork of fibroid tissue. Hence, if we take up the relations of the two with a philosophical desire to see analogies and keep in view the generic common likeness, we may easily look on adenoma and carcinoma as in one category, or essentially the same thing, as indeed Rindfleisch does, but it is a serious practical misfortune if we are obliged to weaken the distinction between innocent and malignant tumours; a practical aim was before those who commenced the study. It was not to reach an abstract knowledge of tumours, but to find new means of discerning their clinical tendencies that the microscope was called in aid. Our object must be to keep as sharply clear as we can the distinctions which enable us to form true views of the future of the case under treatment; and if the end of microscopic investigation is to make us think that adenoma is anywhere about the same as carcinoma, then so much the worse for the microscope; we cannot be guided by it. This result might have been expected. No doubt with a binocular microscope on his eyes, so that he could see no distinctions that were not microscopic, a farmer would not know his swedes from his turnips by the look of them, and would be worse off in proportion as his microscope was powerful. Microscopic power increases of late years with an ever increasingly microscopic gain. The effect of histology in the particular question we are considering is negative; it sees through and through a number of criteria, which were practically useful without it in distinguishing deadly from innocent growths of the breast, and thus so dazzles us that by its aid we cannot any longer tell carcinoma from chronic mammary tumour as people formerly could, and as we can ourselves if we put away the microscope.

The difference between adenoma and carcinoma is commonly the difference between life and death, and as the microscope has latterly plainly tended to confuse these kinds of tumour, we shall not in our descriptions of them recommend to you at all an exclusively histological view, but rather get you to consider them in a general way, using all the means which your senses furnish towards knowing the several kinds.

We shall describe the tumours of the breast as—1st, simple cysts due to dilatation of the ducts; 2nd, solid growths of structure similar to that of the mamma, or growths of sarcoma and myxoma mingled more or less with mammary structure; 3rd, tumours of the same composition as the last with cysts into which often project polypoid masses of the tumour of various shapes; 4th, carcinoma in its several forms; 5th, other occasional tumours, as certain large cysts, enchondroma, hydatids, &c.

Cystic dilatation of ducts, or galactocele.—Sometimes one of the ducts dilates, and the milk collects until a large tumour is formed; when opened, the fluid escapes, or, as in this specimen of Mr Birkett's, a change may take place in it, whereby the fluid parts are absorbed, and the solid mass of casein remaining is found in the cavity. There may also be a general dilatation and disease of many ducts, as you see in this specimen, which required removal.

Adenoma—Chronic mammary tumour.—By this we mean a tumour resembling the natural tissue of the breast. This is not frequent in a pure form, but it is found usually combined with sarcomatous or myxomatous substance. It arises in one or several lobes of the gland, sometimes so superficially that the whole gland appears to be free though pushed aside or spread over the tumour, which may be lobed, or nearly globular. There is a capsule composed of connective tissue compressed by the tumour. Section shows a subpellucid succulent mass almost homogeneous, and so differing from the more dangerous sarcoma and myxoma, which have a conglomerate, discoloured appearance.

The tissue, examined by the microscope, is not so perfect as that of the normal breast, for the larger ducts are mostly wanting, it being very rare to find any fresh formation of them, but their cæcal terminations are seen as in the healthy gland, and lined with epithelium in the same manner; it is these you must look for when you are examining a suspected specimen by the microscope, and you will soon discover the rounded terminations as you see the healthy gland drawn in your books.

If expert in the use of the instrument, you can generally see the blood-vessels and lymphatic spaces between the acini, and you will see that the glandular epithelium is just like ordinary mammary glandular epithelium in the active gland. Also, generally, the lumen of the follicle is preserved (we have repeatedly seen this in well-marked adenomatous tumours, although we have heard it denied). The differences from cancer which such tumours show sum up, perhaps, best in this, that these mammary tumours are exactly like active mammary gland and cancer is not. The microscope brings out the minuter differences as the naked eye shows the larger. In carcinoma you do not see with the microscope well-defined acini with regular vessels in their walls and proper glandular epithelium lining their cavities, as you do in these growths; also by the naked eye the following appearances distinguish them from cancers; their defined surface enclosed in a capsule of compressed tissue, their structure pellucid and homogeneous, the substance not retracting on section, no milky juice yielded when the surface is scraped, and no infection of parts around or of the lymphatic glands.

Adeno-sarcoma—Adeno-myxoma.—We have just described adenoma as a development of new glandular substance in the form of a tumour. Such new glandular substance includes several components, in particular the acini, the interstitial tissue between them, blood-vessels, and lymphatics; now, in some cases masses of new growth appear in the breast, which when you examine you find to contain a variable proportion of glandular acini with their characteristic epithelium, but the interstitial substance between these acini is in great excess, and instead of being simple connective tissue, has the structure of sarcoma—that is, it is composed of spindle or round cells with intercellular substance. When you see this structure under the microscope and find a few glandular elements and much sarcomatous interval, the whole arising from a lobe or two of the original gland, or perhaps from a larger part of it, the question naturally arises whether the acini are not persistent elements of the normal gland and the sarcoma the only new matter. When the proportion of gland is small this is probably the case, but we have seen several examples wherein along with sarcoma there was an evident excess or hypertrophy of the glandular acini. On the other hand, no doubt can exist that in many cases the acini represent only persistence of the natural elements. Such growths are called adeno-sarcoma,

Similarly you will find other cases in which the like compounding of gland tissue with myxomatous structure occurs in varying proportions. Such tumours are called adeno-myxoma. The appearance to the naked eye in such tumours is not unlike that in simple adenoma, but they do not show the glandular appearance, being more uniform and more opaque. Generally, too, the tumour is more divided into lobes, and the lobes themselves differ in hue and consistency. The glutinous touch and gelatinous consistence of the myxomatous form distinguish it from sarcoma.

Sarcoma.—In some cases you find large tumours of characteristic sarcomatous structure in the breast—Paget's recurrent fibroid, the composition of these being quite typical.

They are more distinctly fleshy and subopaque than adeno-sarcoma, but the microscope is required to distinguish with certainty between them. These tumours are likely to recur after removal, as in the instances before you; the subsequent growths are generally softer than the first, and it has been said that a change of type appeared after frequent recurrence, so that the structure was like carcinoma; but this may have been alveolar sarcoma.

Myxoma.—Likewise in some cases myxoma arises in the breast, quite typical in its character. It may be found in the form of a distinct pendulous tumour, or an encapsulated growth, pushing the breast aside or including much of the mamma. In section a myxomatous tumour generally appears composed of lobes of different consistence and colour, but all more or less gelatinous. You can often trace all degrees of softening, to the formation of cysts. At other times the consistence is fleshy. We have met with examples in which whitish richly cellular material surrounded the blood-vessels for some little distance, being then enclosed by a defined surface, so that the blood-vessels formed thick, white, wormlike coils in a clearer and less consistent material, such as we have described under the name of peri-angioma. Soft myxomata of the breast are likely to recur.

Fibroma.—Tumours of well-developed fibrous tissue occur sometimes in the breast, and may reach a large size. These are innocent, and form rounded, movable, massive growths. They must not be confused with atrophic carcinoma, which sometimes shows little more than fibrous tissue.

Cystic adenoma—Cystic sarcoma, Cystic myxoma, &c., ad libitum, are a set of terms which express the simple fact that cysts may develop in any of the forms of tumour to which allusion is made in the name. Such cysts are at least of two distinct kinds. Sometimes they are dilated ducts; sometimes they are formed by a solution of the tissue; perhaps they also arise in other ways, as by hæmorrhage into the soft mass, by widening of casual interstitial spaces through accumulation of fluid, &c. The cysts caused by dilatation of the ducts are generally not far from the nipple. A duct may be traceable into one or other of them. Further, the microscope shows that they are lined by cylindrical epithelium. Such cysts are often of large size and numerous, but they are usually flattened into fissures, and on stretching them open you find spaces into which polypoid, laminated, or cauliflower-like masses project from the wall. They are usually described as arising by obstruction and distension of the ducts. From this you would expect them to be hollow spaces instead of fissures, as we have described. To explain their being closed, and yet due to distension, it is generally supposed that they once were uniformly distended, but that the polypoid masses grew into and closed them; there may be some truth in this, but we believe that the assumption of their ever being dilated into wide spaces and subsequently filled up is incorrect. You remember the enormous surface which the prostatic urethra shows in an hypertrophied prostate, forming a fissure of many square inches superficies. This is caused by the growth of the superficial urethral tissues, which *share in the formative activity*, while they retain connection with the growing mass of the prostate, and so while they increase are drawn and thrust into such shapes as the new developments may compel by their pressure and traction. In a similar way, partly by growth of the duct-surface and partly by its being drawn and thrust by the irregular pressure of the unequally but greatly enlarging tumour tissues around, the surface of the duct is expanded and stretched about, around, between, and otherwise among the ever increasing lobe-like masses which began in its walls, until it becomes a great irregular fissure amongst these lobes, such that when you stretch the fissure they appear to have grown into a space. This is, we believe, the mode in which the fissure-like cysts arise; they never were hollow spaces, but grew as chinks and clefts. They are often very numerous, so that the section of such a tumour has been well compared to the cross cut of the heart of a cabbage. If we inquire why in some cases the ducts should thus form cysts and in others should not do so, we discover, in answer, a certain analogy in the behaviour of the surface of the skin and mucous membranes in the formation of tubers, condylomata, and papillomata; for in tubers, which form in the deeper texture, the surface of the skin grows little; in condylomata, which form in more superficial texture, the surface becomes complicated, and in papillomata, which form from the very surface, it becomes richly and intricately complex, thus showing that when tumour growth implicates a natural tegument close to the surface there is no limit to the extension of that surface which it may bring about. If, then, we suppose that in these cystic growths the cause of the tumour implicates the surface of the ducts, expanding it like papilloma does the skin, while it still retains its epithelial surface. v can understand how such cysts may arise when in other cas surface of the duct not being implicated, no such expansion of the duct occurs. As we have already said, such polypoid development in chinks and fissures occurs both in adenoma, sarcoma, myxoma, and intermediate forms, giving rise to cystic varieties of those forms.

Besides the cysts thus developed from the ducts, you meet with others plainly produced by solution of the tumour tissue, especially in myxoma. Hæmorrhagic cysts are more frequent in carcinomatous and other malignant tumours; besides these you occasionally meet with small globular serous cysts, apparently caused by distension of the glandular acini or of interstitial spaces. Such cysts may be found in the mamma without any tumour, or, when a tumour is present, they may be at some distance from that tumour.

Carcinoma.—As we have before said, this form of disease depends mainly for its physical characters on those of the organ wherein it is developed, and so it follows that, in a part which, like the mamma, contains much fibrous tissue, the cancer is of the scirrhous kind; and it is most probable that its hardness is due merely to the fibrous network belonging to the original structure of the gland itself. Thus it is that if a soft or encephaloid cancer of the breast be met with, it is only when the tumour has grown to enormous size, so that the new soft cellular elements greatly preponderate, or else when a secondary tumour has arisen after excision of the breast, so that the growth has no old fibrous tissue in it, and the stroma or matrix of the growth is a new formation, and is of a comparatively slight kind. In these secondary growths, however, the tumours are at their commencement very hard, since they spring up in the skin around the cicatrix, and this consists of a dense fibrous structure. In ordinary *carcinoma* of the breast the tumour is hard, and the cut surface shows a fibrous-looking fabric.

The leading characters of such a cancer are, first, its tendency to contract, exerting tension on the parts around, drawing in the nipple and skin in a puckered way, and in section showing tough fibrous cords running into the fat around. The same internal tension is shown by the shallow cupping of the surface on section, which is a remarkable feature. The next leading character is the tendency of the elements to degenerate, shown by the whitish, opaque, curdy, or even cheesy patches, minutely mottling the surface, which elsewhere is opaque but creamy or milky with a pinkish cast, the whole minutely reticulated with faintly bluish lines of fibrous texture. Again, other characters are the implication of the glands and the continuous infection of the skin and other tissues, so that they are incorporated with and not merely stretched over the tumour.

Carcinoma is distinguished into several kinds according to the character and quantity of the cellular element. Firstly. *Medullary*, where the cellular growth is free and the stroma therefore proportionately soft. This kind is liable to hæmorrhage and degeneration; it is rare in the breast, and may grow to a large size. Secondly. Carcinoma *simplex* is the ordinary form; its characters are those above described, the consistence being about that of a raw potato. Thirdly. Scirrhus or *atrophic* cancer shows a great proportion of the fibrous substance. The nests of the cells being very few, and often being in a state of degeneration, which may be so complete that the whole looks like fibrous tissue, thus giving rise to accounts of fibrous cancer. Some distinguish between scirrhus and atrophic cancer; cases with small cells and no fatty degeneration of these, but with a great preponderance of fibre going by the former name, while the degenerating forms are called atrophic. A *tubular* variety is spoken of by Billroth in which the cells run in tubular spaces thought to be lymphatics.

The original starting-point of the disease is a question on which opinions are extremely various. Virchow considered its malignancy to depend on a growth of epithelial elements from connective corpuscles. The "*heterologous*" character of such a development on his view would explain the malignancy. The older opinion of Rokitansky, which still has distinguished expounders such as Thiersch, is that the epithelial elements are developments of the natural glandular epithelium, Rokitansky thinking this to be through the local influence of the glandular neighbourhood upon fluids effused, while Thiersch considers it a hypertrophic solid outgrowth of the normal gland. Rindfleisch adopts an intermediate position, and describes the gland-follicles as at first proliferating into cancer, and afterwards affecting the interstitial substance with the action; at the same time he leans a good deal to a rather distinct view especially favoured by Köster and Billroth, in which it is thought that a principal seat of the activity of carcinoma is the endothelium of the lymphatics. Thus you will see that the prevailing solidism gives a start in life to abundant conflicting theories. If any other tissue were present no doubt some one would soon advance it as the origin of cancer. According to our own observations all the tissues are alike implicated, but the earliest stage appears to be the formation of great groups of cells in the place of individual connective-tissue corpuscles. Still there can be no doubt that in the further progress of the cancerous change the epithelial elements are implicated. Close around the cancer you often see crowds of small corpuscles; some think these are young cancer cells, but they may be due to inflammatory infiltration.

Cancerous tumours may remain for many years without causing inconvenience. In such cases the disease is of the atrophic or scirrhous kind. Sometimes, but very rarely, the cancer has been known to undergo calcareous change. In a few cases cancer has been found associated with a great cyst, the cancer forming only a patch on one side of it. Such cases have been of slow development. Sometimes cancers do not return after removal, but in most instances the whole history endures for only a year or two; the disease returns on removal, and after death the internal organs are commonly found affected; mostly the liver, lungs, and sometimes the bones of the skull and dura mater. The disease may be propagated through the body as independent deposits, but very often the interior is affected by a gradual extension of the cancer through the walls of the chest to the pleura, or along the cervical to the mediastinal glands, thus reaching the interior of the body: on several occasions, however, we have examined persons where the local disease had caused death by exhaustion, and no cancer was found internally, although the organs may have been otherwise degenerated. As we have before said, the general atrophy of the system so often associated with cancer is due immediately to interference with the function of some important organ, and thus it is that in these cases of cancer of the breast the bodies are often very fat, affording a strong contrast to the wasted state of those dead from cancer of the stomach.

Colloid carcinoma.—This is not frequent, yet not very rare. It usually appears as a colloid transformation of the cells of a carcinoma with great enlargement of the containing spaces until the reticulum of the cancer is reduced to fine threads, and the cells to a gluey material containing a few corpuscles. Some examples called colloid have proved on examination to be glutinous and soft myxomata.

Enchondroma, in a few instances, has been met with in the breast.

Lipoma may occur about the gland, and simulate tumours within it.

Hydatid.—We need not inform you that the hydatid disease of the breast, spoken of by older writers, is what we know as cysto-sarcoma, and that real hydatid is a very rare disease. We have seen, however, several such cases; in this specimen you see the parent hydatid or echinococcus, filled with smaller ones.

UTERO-GESTATION

We shall not say much on this subject, as it is so fully treated of in the obstetric lectures; but there are certain parts of it which are constantly coming before the morbid anatomist, and upon which his department has thrown great light; for example, abortions, which formerly were thought to arise from some accidental circumstance, are now known to arise, in the majority of instances, from disease of the ovum.

OVUM

We have repeatedly said, that most of the morbid actions in the body are chronic, and the same statement applies to abortion. It was formerly thought that this was always due to some violent exertion or excitement on the part of the mother, which caused the uterus to discharge its contents prematurely; but an examination of the aborted ovum generally shows a change in it, which must have been progressing for a considerable period. In some cases it is probable that from the time of conception the ovum is so slightly blighted that no embryo appears, while yet its membranes may continue to grow. You will find all degrees and varieties of imperfection of the embryo in different cases.

You will often, however, have ova shown you which appear healthy; when this is so the deciduous membrane is perhaps seen on the surface, but it may be retained and cast off afterwards, so that the chorion then forms the outside of the aborted ovum; or a mass resembling a mere blood-clot may be discharged, its separation being due to sanguineous effusion in the deciduous membrane, which has caused a separation of the chorion, and thus the ovum may be found in the clot. At a later period the cause of abortion will generally be found in a *disease of the chorion;* for when this is gravely affected, containing as it does the vascular loops through which the nourishment is conveyed from the mother to the fœtus, the ovum necessarily drops off. That the disease is due in the first place to some constitutional defect on the part of the mother is suggested by the repeated occurrence of abortion in the same patient; no doubt a chronic inflammatory state of the interior of the uterus would be sufficient to cause it. When the chorion is examined under these circumstances its villi are found to be of a white colour; if some of them are placed under the microscope they appear quite opaque from the presence of crowded fat-globules and granules, the natural cellular structure having disappeared. In some of these cases also you will find the villi dilated into small cysts, approaching in character the form of disease we shall next describe. These changes may be found in the chorion at a very early period. Thus, we have in two or three instances found a spherical mass of the diameter of a florin, which was quite white on its villous surface, while the interior was much discoloured with hæmatoidin pigment, no trace of the embryo being discoverable. Such cases are not easy to understand; they suggest that some bleeding occurred very early in the formation of the embryo, stopping its development, the ovum being retained until the embryo wasted away. In one of these cases the villi of the chorion showed commencing cystic degeneration—a fact that favours the belief that the cystic chorion develops after the death of the embryo.

Cystic Disease of the Chorion is a very remarkable affection; it was formerly called hydatid disease of the uterus, for it was supposed that the cysts were hydatids, and grew in the uterus; indeed, no doubt it would often be difficult to ascertain their source, so that when, after a supposed pregnancy, the woman was about to be delivered, and instead of a fœtus hundreds of bladders or cysts were expelled, it was natural to suppose that these were either parasites or a multitude of human ova. But very often the whole came away in a mass, and then their connection with the membranes was seen, as in these examples. You observe that they resemble large bunches of grapes, and are of various dimensions, though the cysts never reach any great size. If you examine them, you will find they are formed by dilatations of the villi, as in these drawings; not that there is merely one at the end of each villus, but you will see a succession of the cysts, one above another, resembling a string of beads. This has suggested an origin by the budding of one from the other, a supposition which gave the title "proliferous cysts" to these bodies; but Dr Braxton Hicks has contested this belief, and is of opinion that the connection of them in rows arises through the cystic change coming upon many different points in the course of the long vascular tufts. Even where you see little buds starting from the cysts, like one cactus leaf from another, he thinks these buds did not grow after the cystic change, but had already formed when overtaken by it.

As to the pathological structure of these bodies the word cyst scarcely applies to them; for though very full of fluid, they have a solid meshwork. This consists of delicate fibrils with a few stellate cells, while the fluid which distends the "cyst" contains mucus, as was shown by Virchow, who considers that the mucous "Wharton's jelly" of the umbilical cord is continuous in the placenta with the substance of the villi. On these grounds he classes the "cysts" as myxomata, and, indeed, considers them as excellent types of the class. The same observer found in a full-grown placenta a large lobulated mass of a like myxomatous structure.

How the cystic change comes about is one of the most strongly contested questions of pathology. The usual view at present is that there is first a primary disease of the placenta, which itself is the cause of the death of the embryo or fœtus; Virchow suggests that it may arise from endometritis, or otherwise from some evil influence of the maternal blood or tissues on the ovum. On the other hand, Dr Graily Hewitt supports the older view that the change is really a dropsy of the chorion villi, due to death of the embryo or fœtus, and perhaps showing itself long after that occurrence. In favour of this view it is true that the dead fœtus is very rarely to be found in such cases, and in considering their history they are often traceable to partial abortion about the eighth week, while similar changes may appear in portions of placenta retained after the full term. This seems the more probable view, and yet several circumstances cast doubt upon it; especially it is urged by Virchow that long retention of the placenta of a *dead fœtus* is not sufficient explanation of the change; for in extra-uterine pregnancy, and in other conditions where the placenta remains long in the body, it does not undergo these changes; indeed, the retained placenta is known to assume a compressed form; again, where the cystic change is supposed to have occurred in retained placentæ after childbirth, better observation has proved the cysts to belong to an abortive twin fœtus or a superfœtation; while, lastly, a partial cystic change has been found in the placenta at full term in the presence of the living fœtus. This is the most cogent fact of all; thus the question must at present be left uncertain. It is said that the affection implicates the whole surface of the chorion if it commence before the formation of the placenta; if afterwards, it is limited to the placental region. Virchow mentions a case where the placental attachment was the only part unaffected.

The *amnion* may be diseased, but this has more to do with practical obstetrics; an excess of fluid is called dropsy of the amnion. It has nothing to do with dropsy either of the mother or fœtus, but it appears to depend on local causes. It may affect one of twins. Sometimes blood may be found within the amnion. More rarely an adhesion of it to the fœtus has been found. These adhesions are of great importance as causes of malformation. Cord-like bands may constrict and amputate the limbs, and local adhesions of the fœtus to the amniotic surface may cause, or at least go with, non-development of the affected part, as of one side of the head, &c.

Extra-uterine Pregnancy.—Sometimes, instead of the impregnated ovum arriving safely at the uterus, it is delayed in the Fallopian tube, where it undergoes development, either in the portion of the tube that is in the uterine wall, in which case it is called "*interstitial*," or else in the free part of the tube, when it is called "*tubal*," or it may be attached only to the fimbriæ of the tube; or it stays free in the abdominal cavity, and fails to enter the tube at all, when it is called *abdominal*. It is even said that the fœtation has happened in the ovarium itself, but we have never witnessed an example of it, unless, indeed, the hairs, teeth, &c., sometimes found in ovarian cysts, might be the result of delayed impregnated ova—an opinion that cannot be maintained, since similar growths are found in other parts. We have several times noticed an adhesion of the affected tube to the omentum, &c., as if it had been impeded in its function by this. We have one example of interstitial pregnancy, but of the tubular kind at least a dozen. You will see the embryo is in all stages of development in the several cases; in some death of the patient has occurred as early as the sixth week, while in others the child appears almost full grown. In one curious case two fœtuses of different ages have been found attached to one tube. In other cases abdominal and uterine pregnancy have occurred together and reached their full term.

When the ovum is arrested in the tube all its parts become fully formed, as in the uterus; a good chorion surrounds it, which gathers nourishment from the tube, this having a large supply of vessels from the spermatic arteries. It has still to be determined in what way the connection is formed; it was the opinion of Dr Robert Lee that a little whitish soft material, which we find enveloping the chorion, is in reality a decidua, and from this he argued that the deciduous membrane, in all cases of pregnancy, is a part of the fœtal structure rather than the uterine. It is remarkable, however, that these cases of extra-uterine fœtation have been taken as a corroborative proof that the decidua is formed from the surface of the uterus (if it be not, indeed, its altered mucous membrane), for you will see in all these examples that the uterus is lined by a thick, pulpy substance, which has a cribriform appearance, being pierced with a number of holes corresponding to the tubular glands of the organ. This is generally looked upon as the deciduous membrane developed in the uterus whilst the ovum is growing without; the uterus itself, you see, is enlarged, although containing nothing. It is even now a contested question to what extent the uterine mucous membrane is thrown off in the formation of the decidua and renewed in the course of pregnancy, some holding that it is quite exfoliated, as Cruveilhier thought. Others hold that there is a partial exfoliation about the third or fourth month, while in the later months the membrane grows again beneath, covering in the muscular tissue, so that this is not exposed at birth, but is lined with a thin, new mucous membrane; most good authorities agree with this opinion, we believe,

It is a remarkable circumstance that in several of our specimens the corpus luteum is on the opposite side of the body to the ovum. This would suggest that the Fallopian tube must have sent its fimbriated extremity over to the other ovary in order to seize the ovum; of the possibility of this we are not certain. Or the ovum must have passed into the uterus, and then again made its exit on the opposite side; unless it be that the development of this body occurs indifferently in any Graafian follicle which discharges its ovum about the time of impregnation, whether it be the one actually implicated or no.

In most cases, when the ovum has reached any size, and the chorion has begun to collect into one mass to constitute the placenta, the tube bursts, and death takes place very rapidly by hæmorrhage; if, however, only a slight lesion should occur, an inflammatory process may take place, causing an adhesion of the containing sac to surrounding parts, while development goes on. When the full period of pregnancy is reached, and the fœtus is unable to escape (the same occurs if it should die prematurely), it acts as a foreign body; adhesions are formed to surrounding parts, suppuration occurs, and the bones and other portions escape by openings in the abdominal walls or by the rectum; in one case, at the end of gestation, a woman was taken with the usual signs of labour, which passed off in a week; her health then began to fail, abdominal pains distressing her; after three years an abscess broke into the rectum, and some fœtal bones escaped; these continued to come away for another three years, when the woman recovered. Sometimes, however, the fœtus has dried up or mummified, and remained quiet for several years; in this specimen you see a mass of bones which were long retained in the abdomen; or adhesions caused by it have strangulated the bowel.

PLACENTA

As the diseases of the ovum, and especially of the chorion, have to do with abortion, so do the diseases of the placenta constitute, very frequently, the causes of miscarriage. Sometimes the changes in the placenta appear to be merely a premature condition of what is natural at the end of the ninth month of pregnancy; at this period the placenta separates from the uterus like fruit from a tree, having lived its natural period of life, so that it is suffering the same process of decay which the whole body does in old age; thus an earthy matter is constantly formed in it, and the *placental tufts undergo a fatty degeneration*. This has been disputed, yet the occurrence of some degree of fatty degeneration here is so constant as to leave no reasonable doubt that it is natural at full term. But if this change should occur prematurely, a miscarriage is the result, and then, if portions of the placenta be put under the microscope, the degenerative changes may be discovered, or the morbid deposit may even be seen as white specks by the naked eye. Generally, however, during the gradual separation of the fœtal and maternal structures, some oozing of blood takes place, and then, besides much that escapes externally, the placenta may be found occupied by large coagula—a condition to which the name "apoplexy of the placenta" has been given. As, however, the separation is gradual, the blood has time to undergo various changes, the colouring matter is taken up, and the fibrin is left. It may be, however, as Virchow urges, that such fibrinous masses are more frequently the result of coagulation of blood in the maternal part of the placenta than of actual apoplexy. In many of our specimens the placenta, instead of being spongy and soft, is hard, and of a yellow-white colour; the structure within is firm, and the vessels and villi are with difficulty separable, being united into one compact mass. This state is, however, present only in parts, and thus several distinctly circumscribed lumps or tumours are formed within it. If these be carefully examined by the microscope after tearing up a portion of the tissue, the villi will be found charged with granular and fatty matter, and the fibrillated and granular material of old blood clot will also be seen between them. You will notice in the museum that many of these diseased placentas were called tuberculous, for the reason that all unorganizable or amorphous deposit formerly received this name; but the pale, solid matter is merely altered blood. Indeed, it is universally agreed that tubercle or cancer cannot, or at least does not, occur in the placenta. The effusion is probably due to a diseased condition of the natural structures, but whether this, indeed, be owing to any delicacy of constitution, of a scrofulous or other kind, on the part of the mother, is another question. Subsequently, owing to the resemblance of this adventitious matter to lymph, it was thought that the consolidation of the placenta corresponds with the similar condition of inflamed lung, and thus the term *hepatization* was given to it. In most cases, however, there is no proof whatever that the lymph-like matter is an inflammatory product; and yet inflammation of the placenta, or *placentitis*, does occur, and by it an exudation of lymph takes place which binds the placenta to the uterus. This is the cause of those rare cases of adherent placenta which some attribute to falls or injuries during pregnancy. It is also very common to find calcareous degeneration of the placenta even to a considerable extent; this is probably a truly morbid condition, and yet lower degrees of it at least are compatible with apparently sound health of the fœtus.

Other morbid conditions of the placenta, referring more to obstetrics, will be pointed out to you in the midwifery lectures; such as the effusion of blood between it and the uterus; also its malposition in the uterus, as placenta prævia, &c.

UMBILICAL CORD

The various affections of the umbilical cord will also be explained to you; such as disease of the blood-vessels, or their plugging; œdema of the cord; knots in it, &c.

FŒTUS

The diseases of the fœtus have of late years been regarded with great interest—not only in reference to any constitutional cachexia to which they may give rise, but as originating those early organic changes which we are in the habit of calling malformations; and when we speak of disease we do not use it in that general sense signifying a mere deviation from the healthy standard, but, in most cases, as a positive malady, such as occurs in extra-uterine life; as, for example, a local inflammation, or a rheumatism. Many malformations, however, must still be ascribed to aberrations in the developmental power, and this is especially true of such malformations as occur in the mesian line from an imperfect fusion of the two sides of the body, such as are seen in cleft palate, extroversion of the bladder, &c., as well as others which arise from excess of development.

These malformations we have already sufficiently spoken of in their respective sections, and we shall now just allude to some other abnormalities special to the fœtus which did not come under the descriptions already given.

A very remarkable set of malformations are those ascribed to spontaneous amputations *in utero*, supposed to be caused by the umbilical cord becoming twisted as a ligature around them. The effect of such twisting could hardly be a complete severance of the limb, for such tight constriction would probably stop the circulation in the cord and so lead to the death of the fœtus. But it has been shown to produce grooves on the limb affected, and it may, by stopping the supply of blood, prevent the growth of the distal portion so as to reduce it to the dimensions of a mere bud on the end of the proximal part. This is probably the explanation of the bud-like growths from the ends of amputated stumps which have been thought to be attempts at reproduction of the lost part.

The actual severance of the limbs has been explained by Dr Montgomery as due to constriction by bands within the amnion; numerous observations have confirmed his view; the separated part, however, is generally not found, but the amniotic liquid is then turbid in some cases, suggesting that it may have disintegrated. A more probable cause of amputation is constriction by the cicatrix of a cutaneous ulcer. Such ulcers have been occasionally met with in the fœtal integuments.

Intra-uterine fracture by violence is another cause that has been thought capable of causing amputation, but it appears very improbable that the yielding tissues of the fœtus would be broken by an injury that did not produce abortion. Other cases are unexplained, and some think then that amniotic strings which did the mischief may have disappeared. In cases of amputation you sometimes see signs of a scar on the end of the stump. But there are a series of cases where the limbs are cut short that do not admit of explanation by supposed amputation—cases, that is, where the deficiencies are symmetrical and affect many joints. In the case from which these models were taken, and in which the hands had lost many fingers, the losses corresponding on the two sides, the man said the malformation was hereditary in his family for many generations; he said, too, that the deficialways happened in the short members of his family, while those with perfect limbs were always tall, some of them being in the life guards. Partial development of a limb, however, must be accounted for on other principles, especially when the disorder follows some anatomical district, as, for example, when the fibula and toes on the outer side of the foot are deficient; withering of a limb may arise from paralysis; and in such a case we have seen an atrophy of the opposite side of the brain as the cause; in a similar case which we have already mentioned, where death occurred during birth, an effusion of blood had taken place in the brain, and it was directly traceable to a fall the mother had suffered during pregnancy.

With respect to the *heart*, the various malformations, though so different in kind, can be for the most part well understood and classified on the supposition of some endocarditis during fœtal life; the proof, however, that the fœtus has rheumatic attacks is still wanting, although in one instance of malformed heart there is a history of the mother having rheumatism during gestation.

We have already spoken of *brainless* infants, and cases where from a hole in the skull a part of the brain or the membranes escape, constituting *encephalocele* and *hydrencephalocele;* also of *spina bifida*, and its association with clubfoot. But besides such malformations, the brain and spinal cord in the fœtus are often found *diseased*. Virchow describes a congenital encephalitis; the disorder has the character of white softening—that is, you find in it granule cells, while the tubular tissue is broken down, its fibrils being sometimes separated into short, rice-grain-shaped bodies, which we have frequently seen. The evidence of its inflammatory nature is not more conclusive than in the white softening of the aged; but Virchow maintains that view on the ground that such white change coexists with inflammatory processes in the kidney and elsewhere; also that it is often caused by scarlatina, smallpox, and syphilis. Some degree of this condition is common in syphilitic infants, and it may be found at any time during the first few months of life; its signification in explaining malformations and imperfections in the development of the limbs is evident enough.

In coming next to the *skin*, we find here specimens which we have not yet alluded to, and amongst them are these three remarkable examples which go by the name of harlequin fœtus, or skin-bound fœtus, or *intra-uterine ichthyosis*. They are not very common, so that our museum may be considered rich in such specimens. They are all at full term; the skin is covered with fissures, which, crossing in all directions, give the resemblance to a harlequin. The impression which is first conveyed to your mind by looking at them is, that the skin had ceased to grow at a certain period, while the tissues within, continuing to increase, had caused distension even to bursting, and thus the integument is cracked and fissured on the most prominent parts of the body. As we do not know that they have ever been described, we will point out more in detail some of the peculiarities which are seen in this specimen. The fœtus is of moderate size, fully developed, and the skin covered with fissures; these are more numerous in front than behind; the lines have a general tendency to be horizontal around the body and abdomen; on the chest they run in oblique directions, producing lozenge-shaped spaces; on the extremities their course is transversely around the limbs; in those parts where the body is flexed, the fissures are just in the direction which would follow from the stretching. The face is fissured in all directions; on the head the cracks pass from back to front; on each side of the body the transverse lines are intersected by wide longitudinal ones. All the fissures have an appearance of having been caused by stretching; in some parts merely the cuticle is affected, but in the deeper fissures the true skin is torn, and you see fibres passing from side to side, just as would be produced by a forcible separation of the skin. In one or two places it appears as if the cuticle had been destroyed and again repaired. The fissures are about one eighth of an inch wide. The eyes are perfect, but the eyelids fixed back to the forehead, so that the conjunctival surface is exposed; there are no eyelashes; the nose is not much altered, the mouth is kept open by rigidity of the skin around, which is as hard as cartilage, and forms a complete rim to it; there is no external ear; there is hair on the head; the fingers and toes are perfect, but are drawn together by contraction of the skin.

There are also other cutaneous affections which may occur in the fœtus: for example, you are all constantly meeting with cases of syphilis where the cuticle peels off; and here is a fœtus which was the subject of smallpox. We just now mentioned to you the probable effects of cutaneous ulcers as causing spontaneous amputation *in utero* by their cicatricial contraction.

As regards the *lungs*, we have seen in many instances acute lobular pneumonia in the fœtus at from six months' development to full term. Such pneumonia may be found in the form of lobular grey or white hepatization. It is most frequent in syphilis; white lobular hepatization is thought by some to be characteristic of syphilis in infants. We may remark that we have never seen tubercle, and we believe that congenital tubercle has not been met with.

In the *peritoneum* many important changes occur from fœtal inflammation, and that this is not uncommon was shown many years ago by Dr Simpson, who found it in children born of syphilitic mothers; many of you may have thought the wretched puny condition of such fœtuses was quite sufficient te show their incapacity for life, but in truth there is very often an acute internal inflammati which has probably been the immediate cause of death; thu now examined many syphilitic infants born dead, and in most of these there was evidence of peritonitis; in two, old adhesions between the liver and the diaphragm, and in the others a general inflammation; in two there was also lobular pneumonia. In the adult you may constantly meet with adhesions, when there has been no history of peritonitis since birth, so that they probably have their origin in the fœtal state. It has also been suggested by Dr Simpson whether the malpositions of the intestine are not due to local peritonitis; and there can be little doubt that such is the case, for wherever these are met with, adhesions are always found binding the errant part in its new position; we allude especially to those cases of which we have lately seen three instances, where the ascending colon and cæcum are dragged over to the left side of the body; you may remember that in one case you heard a discussion as to whether this was due to a malformation or to an inflammatory process; but if we consider the altered position to be due to adhesion in fœtal life, both suppositions are true. It has also been thought that the non-descent of the testis may have been due in some cases to peritoneal adhesion, especially where a coil of intestine has been found connected with the internal abdominal ring. Sometimes the intestine is open at the umbilicus, as in a case related in our 'Reports;' this is occasioned by the umbilical vesicle, which in fœtal life is connected with the intestine, not closing as it should do.

The liver is naturally of large size and dark colour in new-born children; it is but little subject to disease in the fœtus, except in congenital syphilis; in this condition it has been found to show gummatous patches. This we have ourselves never seen, but there is another more frequent form of congenital syphilitic hepatitis in which the organ is pale and hard, being elastic and bloodless. The disease is due to the production of new inflammatory matter in the form of organizable round and spindle-celled lymph in the tissue of the liver about the portal veins. By the pressure of this the proper tissue perishes, but very evenly, so that the surface is smooth and the section uniform. The organ becomes remarkably hard and solid, but the affection is not very conspicuous, and may be overlooked unless great care is exercised.

The kidneys are lobulated on the surface. We have spoken of the congenital cystic change occasionally found in them. It is not uncommon to find concretions of uric acid and urates in the pelvis and on the pyramids of the kidney.

Monstrosities.—These may be of various kinds; sometimes mere masses of organic matter, with only a slight resemblance to a human being, may be looked upon simply as imperfect growths. The most remarkable monsters, however, are those where a body exists with some extra portions attached, and very diversified opinions are held as to whether these be due to an excessive germ power or to a union of two ova, of which one only is perfectly developed. In the case, for example, of the Siamese twins, there can be no doubt that two fœtuses are simply united; while in instances of six fingers to a hand the case cannot be looked upon otherwise than as an excessive development of one fœtus; but if an extra limb be found protruding from the body, a difficulty arises as to whether one or two germs were involved. The union of two fœtuses is clear in a case like this, where the bodies are separate, but the heads united. It is evident too that one ovum may be included in another, and thus we might have the case, as in the Chinese model of A-ke (if he be not altogether fabulous), where a well-developed man has the buttocks and lower limbs of another person protruding from his body. It only requires the limbs of such a case to be joined together to produce the three-legged monster which was made a public exhibition of some years ago; the middle limb had ten toes, and the child, which was alive, had double genital organs. A smaller limb is here represented; it was removed by Mr Cock about two years ago; it grew, as you see, from the lower part of the abdomen, and when cut contained a long bone, like the tibia, running through it; the fact that these occur in the median line of the body, suggests that they belong to a second ovum. Sometimes this second ovum does not project in the form of additional limbs, &c., but forms a tumour more or less included in the body; if so it is generally a mere shapeless mass, or has little pretensions to the figure of a human being; as you see in this model of the curious fœtus found attached to the stomach of a boy, and also in these drawings of several other recorded cases. In this jar you see a quantity of bones which were taken from the buttock of a child, and which must have belonged to a twin fœtus. When we come to smaller protuberances from the body, the question arises, as we before said, as to their being portions of a second ovum, or due to excessive power in the single ovum; as, for example, in this curious body, removed from the back of an infant, and which was called a tail.

ON THE ASSOCIATION OF MORBID CONDITIONS

Having considered the structural changes of the various organs separately, we will briefly point out how these are more commonly associated in the bodies which are daily opened in the post-mortem room; and also indicate those morbid changes which we are likely to discover in such diseases as are known by more general names than those which apply to local structural affections. For you must be aware that a simple demonstration of the morbid anatomy of particular parts is only one step towards a knowledge of pathology; while the study of their morbid associations as we find them grouped in actual cases raises us a step considerably higher, so that we believe a thorough acquaintance with the whole of the morbid processes, in any particular case, enabling us to see how one morbid change is related to another, would lead us very close upon its true pathology. Our present means of investigation, however, will not allow us to demonstrate all the various pathological changes occurring in the body; for there are many, especially those in the nervous system, of which we are at present profoundly ignorant. It may be that some of these changes are of a chemical rather than an anatomical nature, affecting the composition of the juices rather than the forms of the solid parts, as appears to be the case in diabetes, &c.; and it may be that some, like the poison *strychnia*, are of a nature related to the vital or nervous force, calling out the nervous force of one part of the body to effect the death of the rest without any anatomical alteration of either. Even when the morbid conditions are widest, we warn you not to take them as ultimate facts, and think that you have arrived at the true pathology in them. For very often the great mass of disease which we find in a body is secondary, only representing the effects of causes that escape our observation, even when the proper connection of the appearances we discover is clearly traceable, which is not always the case. It is this tendency to lose sight of the causes and treat the effects as ultimate which has brought some odium on the science of morbid anatomy; you need not, however, be alarmed at studying it *too much*, for by a thorough knowledge of effects we must be in a better position to apprehend the nature of their causes. Without slight to the study of etiology and nosology, we may hold that the anatomical side of diseases offers us a view of them which we can realise and know, and upon which we can mutually compare our knowledge, while the sciences of the causes and symptoms of diseases are often vague or fragmentary, being too much mixed up with tradition and hearsay and ill-contrived experiment.

General Appearance of a Body.—You may sometimes be required to inspect a body about which you know nothing, as we have constantly to do at this hospital; and therefore you should, in the first place, thoroughly examine the exterior before you begin to look inside. You should first notice that decomposition is sometimes very early in its appearance and sometimes very partial. One of its very earliest signs is often a formation of purple streaks along the courses of the superficial veins; whenever you see these streaks you may know that the inner surfaces of all the vessels, and the interior of the heart, and of the trachea, will be deep red, and you must never think this redness a sign of inflammation under such circumstances. The great parenchymatous organs, liver, spleen, kidneys, &c., will be dusky purplish, and their vessels, perhaps, will contain air. You can always tell such gas from the air of emphysema by its inflammability. These are states which denote blood decomposition, and that is all they are able to show; while they may render an opinion on the previous state of the viscera difficult or worthless. Sooner or later, they all set in, along with decomposition, in all cases, while the blood exuding from the vessels, at the same time and similarly stains the textures in the dependent parts of the body, into which the blood sinks by gravitation. Decomposition then soon declares itself, the abdomen turning green, and becoming distended with gas, the presence of which forces frothy fluid from the mouth and nose. In some cases the abdomen changes to a greenish colour before the blood decomposes. In these cases the decomposition is slower. Although there is no doubt that the reddening of the vessels, &c., which we have described, are due to post-mortem decomposition, yet it does not follow that the conditions of blood favouring such changes do not precede death; indeed, the especially frequent occurrence of these changes in febrile cases would indicate their dependence on a morbid state of the blood. They may often be observed to be strongly pronounced, when other bodies which have lain much longer in the same dead-house show no trace of them. The more advanced stages of decomposition, when greenness extends over the surface, is succeeded by blackness and giving way of the textures which you can better observe in the dissecting room.

In your survey of the body seek carefully for injuries, bruises, or the like, and never forget, in the absence of all other cause of death, to examine the cervical spine for fracture, or the larynx for a foreign body. When the body looks healthy, you may suspect an accidental death, or an acute disease, or one which has proved fatal suddenly; but if it be wasted, you at once discern that a long-standing ailment must have existed, and then it will be well to put the body through a course of physical examination, as you constantly see done here. In chronic disease the emaciation varies in different affections; the most common cause of it is phthisis; but it is not so excessive as when death has occurred from abdominal affection, as of the stomach, mesentery, &c.; in phthisis the face retains a certain amount of plumpness, while in abdominal disease, where actual starvation takes place, the wasting is much more obvious, the temporal muscles are atrophied, and the fat removed from the orbits, so that the eyes will seldom close; the skin too is soft in phthisis, while it is often harsh in the other affections. You may notice too, with advantage, the condition of the hair in these wasted bodies, which always tells a tale of long-standing disease: instead of its presenting the curly crisp appearance of health, it is long or lanky; and this is especially observable in the beard and on the pubes; you may also notice that the hair has grown considerably on the chest in phthisical subjects. We then generally percuss the body, and if we find the chest dull on one side, we obtain a notion of phthisis; if with emaciation, the disease is chronic; if there be not much wasting, it is more acute, or acute upon old; further, if we open the mouth, and again strike the chest, we may often obtain the sound known as the *bruit de pot felé*, generally, but not certainly, indicating a cavity; sometimes we find the chest tympanitic, denoting a *pneumo-thorax*, and thus explaining the sudden death of our patient since we saw him last. In a case which occurred here the other day, where the chest was dull and the body not wasted, and at the same time an herpetic eruption existed on the lip, the diagnosis was *pneumonia*, which turned out to be correct. In those dead of various chronic diseases we may often find that pleuritic effusion or pneumonia has ensued since the last time we were enabled to examine the patient alive. As a good example of the value of physical examination, we will mention a case which occurred here some years ago, and where the diagnosis made after death proved to be correct in every particular. A lad came one Wednesday morning to be admitted, and died in the taking-in room. Before hearing from his friends we proceeded to examine the body. On percussing the chest, the left side was found tympanitic, indicating a pneumo-thorax, but then the body was not sufficiently wasted for phthisis; and this sign suggested, therefore, an acute disease of the lung, which had ended in rupture of the tissue; as this, however, does not occur except in lobular abscess from pyæmia, a local cause was looked for as a source of purulent infection, when all that we discovered was a discharge from the ear, and on placing the fingers on the neck a general swelling was found, and an induration of the jugular vein; it became then tolerably clear that a chronic otitis, with diseased temporal bone, existed; that the lateral sinus had become involved; that phlebitis had extended down the jugular vein to the heart; and so the lung had been affected with abscesses, one of which had burst, and caused the boy's death by pneumo-thorax. On subsequently opening the body, the morbid conditions were found true in every detail, and the whole history was exactly as suspected. Many similar instances we might mention, proving the value of physical examination.

Sometimes we have a person brought from the street, who has died from *pulmonary hæmorrhage*. If the body be wasted, we suspect phthisis; if not, aneurism. In *brain diseases*, the body too is often extremely wasted, but the emaciation, as far as we know, offers nothing characteristic, though, if paralysis or softening of the brain has been present, you may find rigidity or contraction of one particular limb, or the hands clenched, and the thumb drawn towards the palm, and generally a greater proportional wasting of the muscles than in other cases of emaciation; and the abdomen is often remarkably flattened or hollowed out, and you may find irregularity of the pupils. As regards this latter condition, we would say it is only in exceptional cases that anything unnatural is seen in the state of the pupils after death; the rule being that they are symmetrical and of ordinary size, the tendency being at death for the two antagonistic sets of muscles of the iris to neutralise one another. There are various other conditions of the body to be observed; thus, if it be not wasted, and the circumstances of the death suggest some acute affection, we may then look to the skin, and sometimes find a petechial mulberry rash covering it, denoting *typhus fever*. In typhoid fever no rash would be present, except perhaps a slight purpuric mottling of the legs. If there be an unhealthy sore on the body, and especially if a bone be exposed, we may suspect pyæmia, and the suspicion is strengthened if the body have a yellowish tinge.

In acute *abdominal diseases* the distended tympanitic abdomen may suggest the idea of *peritonitis*, particularly if the countenance express what we call abdominal anxiety, still shown after death by the ghastly visage with sunken eye, &c. In cancer of the stomach the wasting is the most extreme that we meet with; various tumours may be felt after death as during life, and attempts at diagnosis should be again made.

If the body be dropsical, this denotes, in by far the majority of cases, some visceral disease (it may accompany mere anæmia and cachexia); if all parts are affected, it shows that the cause is not a local one, but is to be sought in the blood; and we, therefore, if we see the arms, face, scrotum, &c., swollen, pronounce in favour of renal disease. If the lower parts of the body only be dropsical we suppose the dropsy to be due to a mechanical cause in the chest, and then comes the question, Is this a pulmonary or a cardiac obstruction? If there be great lividity of the face, we may suspect bronchitis; but if this be less, and if the skin of the upper part of the body be yellowish, denoting hepatic engorgement, we may be pretty sure that the person has died of morbus cordis. The appearances in such a case are so well marked that you may almost always recognise a cardiac case before you open the body. If the abdomen alone be dropsical, as in *ascites*, a liver disease may be suspected, or a chronic peritoneal affection. If the legs be swollen or only one leg, it may be well to look for a plugging of the femoral vein with ante-mortem clot.

Besides noticing emaciation you should observe whether there be a superabundance of *fat*, for this denotes a morbid condition of the system which accounts for many deaths taking place in persons after slight injuries or surgical operations, such as that for the relief of hernia. You may often notice in persons advanced in age a large amount of fat beneath the integument of the abdomen, as also a large quantity within its cavity, occupying the mesentery, omentum, region of the kidneys, &c.: this is one of the conditions of age; but you may occasionally observe the same in younger persons, especially those who have been much addicted to intemperance, and more particularly to beer-drinking, for spirits, unless indulged in very largely and for a short time so as to prove rapidly fatal, tend to wasting of the body. You will also get accustomed by experience to draw conclusions from the quality of the fat, there being a firm whitish adipose tissue, which denotes health, while there is a yellow oily form, which may be looked upon as morbid. You should also examine the eye for *arcus senilis*, which generally indicates senile or fatty changes throughout the body. The *muscular tissues* should also be noticed; there is the large red muscle denoting a healthy state, and the atrophied pale muscle showing at least a long-continued inactivity, with perhaps also some degree of fatty degeneration.

Rigor mortis should also be looked for; if present it shows that death has not long occurred; but at what time the stiffness disappears we cannot positively say, never having kept any record, for it varies with the mode of death and state of the weather. It may continue under favorable circumstances as long as ten days. It generally begins within five or six, some say eight hours after death; we have repeatedly found it distinctly pronounced, though as yet slight, in the sixth hour. It is said that in persons who have died with absolute suddenness by shots through the heart, &c., the rigidity comes on at once and persists. It is usually affirmed that rigor mortis commences in the muscles of the jaws and extends downward to the neck, trunk, arms, and legs. This order, however, is by no means absolute; we have often found rigor mortis to have come already in the legs when there was none in the arms. The jaw is, however, the part usually first affected. The strength of rigor mortis in any part of the body is in proportion to the power of the muscles that act on the joints there. But in different persons it varies in strength when at its height, with age and with the cause of death. Thus it is scarcely noticeable in infants, and is feeble after prolonged and wasting diseases; it is always most marked in the bodies of robust persons who have died by some rapid disease, such as cholera, &c.

Of the cause of the change little is known; it has lately been ascribed by Kühne to coagulation of the myosine, with loss of elasticity and contractility of the muscular fibre, this at the same time becoming acid. As the acidity passes off, rigor mortis disappears and an alkaline condition and fermentation ensues.

We shall now again shortly review the various sections; partly so that we may ascertain what are the morbid conditions found in the dead body associated with diseases that are called local, but chiefly that we may consider what is found, and what not found, in those affections which are known by general names.

DISEASES OF THE NERVOUS SYSTEM

We have already said, that when you find the *dura mater* affected you must look to the bone as the cause; also a simple meningitis, in which the inflammatory product is *interarachnoid*, is generally secondary to disease of the dura mater, arising from injury or disease of the bone. A simple meningitis, where the inflammatory product is found *beneath* the visceral arachnoid, may occur from injury, and idiopathically from unknown causes; it is not a common affection, and when met with, all other parts of the body may be found healthy. *Tubercular meningitis*, or acute hydrocephalus, we have already described as a peculiar affection of the brain associated with tubercles; so characteristic is the appearance of the affection that it would probably deserve the name, even if you can perceive no tubercles, provided they were present in other parts of the body; we have never yet failed to find them in other parts, and we have yet examined no case where they were not present in the lungs; in some examples the cerebral affection is associated with a general tuberculosis of the whole body; but the brain being the most important organ, all the symptoms which lead to the fatal issue are due to its inflammation. The disease sometimes occurs as an accompaniment of phthisis, though this is the exception.

Cerebritis or acute inflammation of the substance of the brain may be met with as an acute idiopathic affection, and with no other disease in the body, the cause of its occurrence being not at all certain; it is, however, rare. *Chronic cerebritis* and softening is generally associated with disease of the blood-vessels, and may arise in the first place from a rupture of one of them; it is often associated with Bright's disease. *Abscess* of the brain is most frequently connected with disease of the bone, the inflammation having extended from without; or it may be pyæmic, owing to absorption of some poisonous elements into the blood; you should always look for such a cause, for it is Sir W. Gull's belief that a primary cerebral abscess seldom or never occurs. *Tumours* of the brain may be altogether local, and all other parts of the body healthy; but you should look to the several organs to discover similar growths there; death is produced generally by meningitis or else effusion into the ventricles.

Sanguineous apoplexy arises, in the majority of cases, from disease of the blood-vessels, and less frequently from aneurism of the cerebral arteries; in these latter cases the patients are mostly young, the arterial system is healthy, and no disease exists elsewhere. It is very different in ordinary apoplexy, for this is an affection which belongs usually to age, and is associated with degenerative changes in other parts. We have mentioned its most frequent seat in the corpus striatum, or thalamus opticus, in cases accompanied by hemiplegia; if it be rapidly fatal, we suppose that the blood has broken out from one of these parts into the ventricles, and run down to the base of the brain, or that the effusion has occurred within the pons Varolii. The cause is a rupture of a blood-vessel, and as the smaller arteries are so frequently diseased in granular disease of the kidneys whilst there is an hypertrophy of the left ventricle, you will find a large proportion of apoplexies to be associated with granular kidneys. If the joint of the ball of the great toe be examined you may find the chalky cartilage of gout; and thus you obtain evidence of four stages in the causation of death, viz. gout, granular kidneys, hypertrophy of the heart, and lastly rupture of diseased arteries in the brain, at the same time you may find visceral disease elsewhere, and changes denoting old age. Occasionally you may be called to a case where the patient dies insensible, as if suffering from apoplexy, and on examining the brain you find nothing. We have seen a few cases where the post-mortem thus revealed nothing. In two such cases which we watched throughout, the condition during life was marked by quickly recurring "internal convulsions;" the face growing pale and then red with cessation of breathing; in any such case you must consider whether the attack was epileptic; but you must be mindful in such instances to examine minutely the kidney, to see if there be no disease which had caused a cessation of its function. Also examine carefully the cerebral arteries for ante-mortem clots, and test the brain for local softening by a water stream, consider the probability of a "suppressed" attack of grave fever, or of some poisoning, for apoplectiform attacks may even pass off as urticaria. Do not be misled into ascribing death to the watery fluid in the pia arachnoid of a wasted brain; it is better to confess ignorance than to adopt and continue old errors.

Delirium Tremens.—It was formerly thought that the brain is inflamed in this disease, but experience has shown that there is no acute disease in the brain, and therefore if in any case with somewhat similar symptoms an inflammation is present, we should call the case one of meningitis, and not delirium tremens—for in that disease the brain and spinal cord show no obvious states that account immediately for these nervous symptoms—yet the brain usually exhibits a very remarkable degree of the atrophic induration and accompanying changes in the membranes, which we have before described. The other organs of the body, as a rule, present many degenerative changes brought about by the intemperate habits to which the delirium tremens is due; and it is this alteration in the viscera, we think, to which death is owing. It may, indeed, be caused directly through intoxication, and it is said that then a quantity of alcohol is discoverable in the brain itself. But, as a rule, delirium tremens is a recoverable affection, until that time arrives when such changes have occurred in the tissues that improvement is no longer possible. We then find in the body various morbid states; these are mostly of the fatty kind, as alcoholic drinks tend to produce this condition; but in the case of imbibition of distilled spirits a mere fatty *degeneration* ensues, while in beer drinkers there is also a *production* of fat, both on the surface and in the interior of the body; the viscera, especially the liver, may also be loaded with fat, and the heart may show an excess of fat on its surface. In these cases there may be more positive signs of disease, as cirrhosis of the liver, which is large and also fatty in the beer-drinker; granular degeneration of the kidneys, &c.

The arteries are degenerate, and the heart large and coarse. The stomach shows a condition of chronic or subacute catarrhal gastritis—changes, by the way, which are easily overlooked. The textures everywhere are coarse and fibrous, and the bones thickened—effects which have been compared with those observed by Schiff and others after section of the mixed nerves, suggesting that these states arise through alcoholic paralysis. You must not, however, expect to find all these changes together in death by delirium tremens. It appears that the effect of alcohol in any given subject will depend on the relative strength of his several tissues and systems. Thus, we examined the body of a man who said that he had for twenty years taken a bottle of gin every morning before breakfast, and drank in proportion throughout the day, and yet his liver was quite perfect. In another case of a broker ruined by "Black Friday," who ten months after applied for admission, and who stated, apparently truth-fully, that he was temperate until his losses drove him to drink, and who since then had taken little in proportion to the other patient, the liver was almost destroyed by cirrhosis. The truth appears to be that the alcohol acts deleteriously on all the organs and systems, and that they resist in proportion to their natural strength in the particular individual, so that in one with weak kidney and strong brain it causes granular kidney; in another, whose weak point is the liver, cirrhosis; in another, with an excitable brain, delirium tremens; in another, with a delicate stomach, dyspepsia. In short, the alcohol finds the weak organ in a man's constitution, just as heat finds the volatile component of a mixture subjected to it. It is in this way we must understand the occasional denials of the action of alcohol in producing some of its accredited effects on any given organ, the evidence being given by a study of cases of known alcoholic disease of some other organ. Thus, Dr Dickenson argues that alcohol does not affect the kidney because he did not find the kidney frequently diseased in persons who had died from the effect of alcohol on the brain; but the fallacy here is that those who die by the effect on the brain—a small proportion of all drunkards—are people of weak or excitable brain; but those who get granular kidneys—again a small proportion of all drinkers—are those with exceptionably weak or excitable kidneys, and it is not surprising that these weaknesses do not coincide. In other words, Dr Dickenson's numerical method of investigating necessarily overlooks the element of natural predisposition which, we think, cannot be ignored.

Epilepsy.—Some limit this term to cases of periodic fits in otherwise healthy people, such attacks as are universally called *epilepsy*. Others would include in it that larger class of cases where convulsions phenomenally similar are excited secondarily through irritative conditions, such as cerebral tumours, uræmia, &c.; this class of cases is called *eclampsia* by those who limit the term epilepsy to periodic fits. There is no difference in the convulsion in the two cases except that the fits that are secondary to irritation are apt to occur without loss of consciousness, and the part convulsed may point out the seat of the irritation; on the other hand, those that are spontaneous, that is, truly epileptic, always take away consciousness, without convulsion when they are partial at all. Nevertheless, when the convulsions are complete in either kind of case, the phenomena of both are alike.

Whichever you mean by epilepsy it is now generally agreed, as the result of all inquiry, that there is no morbid anatomy of the fit itself, that is, of epilepsy or eclampsia; you may find the kidney disease which caused the uræmia, or you may be able to show some other poison or may find a tumour pressing, or a syphilitic patch on the surface of the brain. These are to the epilepsy like tumour involving the laryngeal nerves would be to a cough, that is, related indirectly, and one would look just as much to the root of the pneumogastrics in the medulla oblongata for the condition of a bad cough, as one would search there for the conditions of an epilepsy.

As to the mode of death in epilepsy, you find a general venous congestion and turgescence, such as is common to all forms of death by asphyxia.

Chorea.—We have examined many fatal cases of chorea; they were generally children, and hence the brain was comparatively soft and fitted close up to the interior of the skull, and its proper membranes were thin and rather difficult to separate. These conditions have been described as morbid anatomy in chorea; they are rather the normal anatomy of the juvenile age of the subjects who die of it. The swollen odontoid process, occasional tubercle, &c., which have been regarded as causing chorea, we have not seen, though our cases have not been without occasional coincidences, but these were evidently accidental. Neither have we seen any microscopic emboli, such as Dr Hughlings Jackson in his interesting hypothesis supposed to exist, when he thought such emboli would make the blood-supply deficient and unequal, and the action of the centres consequently irregular and uncontrollable. We have met with one case of hemiplegia from embolism during chorea; a similar incident happened under Dr Murchison's observation; but there were no discoverable emboli in the small vessels. Chorea is generally recovered from, and, indeed, it is remarkable that no cases so quickly recover as those which are very acute and threatening. This would infer that there can be no important anatomical lesion. We have seen several such rapid recoveries; they are not unfrequent, and are apt to give an undeserved reputation to the last medicine administered.

The only constant morbid appearance in these cases has been in the heart; you know well that very often in chorea a mitral bruit is heard, and the question has not yet been answered whether the cardiac affection is a functional or organic one; it is remarkable, however, that in all the fatal cases of chorea which have come under our own notice a small row of bead-like vegetations has existed on the mitral valve. Occasionally the aortic valves suffer, as in this instance. Death seems due to exhaustion, for the body is generally considerably wasted, and the skin over the joints and prominent parts of the frame is abraded, from the violence of the movements during lifetime.

Tetanus.—We have already described the state of the spinal cord (page 252); as to the rest of the body, it may be said in general terms that it is healthy. For this reason it is, that it was not the rule to examine those dead of this disease, seeing that no result ever came of the inspection. Showing, however, the value of knowing the appearances found in dead bodies, be they only of a negative kind, we may mention a very important circumstance which arose during the trial of Palmer, as to the state of the heart in tetanus, compared with that in death by strychnine; and witnesses were examined as to its condition in this disease, but facts were not forthcoming as they should have been. It was argued that different effects were produced upon the heart by tetanus and by the poison; the latter has been shown to kill by paralysing the muscles of the chest, or by asphyxia, without having any direct influence over the heart, which is not, therefore, found spasmodically contracted; we have specially noticed this point, and have found that there is no constancy in the condition of the heart after death by tetanus. It is usually contracted firmly, as in most cases of rather sudden death, but it is not unfrequently found to be flabby. The right heart and great veins are, however, found gorged with blood, while there is generally but little fibrinous clot in the heart's cavities, as usual in non-inflammatory diseases that are quickly fatal. The lungs are highly congested or gorged with blood, and all the other organs in a similar condition. The brain is quite healthy. The muscles are dark red, and are sometimes found lacerated from the violence of the spasms, especially those of the back, but sometimes the rectus abdominis has been found torn. The general appearance of the body presents no peculiarities, as far as we know; rigor mortis exists at the usual time at which the bodies are opened, but is not greater, as far as we have perceived, than in other cases; whether it comes or departs at any unusual period, we have no facts on which to form any opinion. You should always examine the wounded part, which is generally the hand or foot; for though it is stated that the nerves are healthy, it is possible that, by repeated and more minute investigations, some morbid condition may yet be discovered; and we ourselves, on more than one occasion, have found foreign bodies, as splinters of wood, in the wound.

Hydrophobia.—It was thought by some that this disease is none other than tetanus, but the general belief is opposed to this; and as during life the dread of water is so characteristic of the affection, so after death the mouth, tongue, and pharynx, are the only parts of the body which present a marked morbid condition. This was observed by Morgagni, who describes an inflammation of the trachea, fauces, &c., in a case of hydrophobia. We have examined only two cases of this disease; in these the body was livid, particularly the face, which looked as in death by strangulation. The blood was of a dark colour, and quite fluid, both in the vessels and in the heart; the lungs were much congested, and posterior parts of a very dark colour and soft; the bronchial tubes also congested, and of a dark colour. The kidneys, liver, &c., all congested; and the brain and spinal cord showed no other appearance than congestion, due to the mode of death. The larynx was healthy, but the pharynx presented a very unusual condition, the mucous membrane was swollen; the glands were enlarged and covered with thick secretion, as were all the glands at the back of the tongue. We have already spoken of these glands (page 357).

Insanity.—We already know from the teaching of physiology that the seat of the cause of mental phenomena is in the grey matter of the surface of the brain, and hence, regarding insanity as a disturbance of the functions of that grey matter, we turn with much interest to inquire whether its state after death in insane persons reveals any destructive alterations. Sometimes we find that there are no changes at all; this is especially the case in acute insanity, and the fact is not altogether surprising, since the acute forms of insanity show rather disturbance than abolition of the functions of the brain; and, again, we know by the transient delirium of fever or the insanity of intoxication that a brain whose structure is sound may be disturbed by poisonous states of the blood, &c., and thus we must think it possible that the cause of mania might reside in the fluids—in short, that the maniac might be intoxicated by some spirit of his own production. Again, the history of the development of different minds, often warped by some dominating set of circumstances, as when the habit of suspicion leads to a madness of fear, &c., will suggest an explanation of insanity in a want of balance among the several parts of the mental phenomena; but how could we be surprised at not finding signs of ill-balanced faculties when we are unable to distinguish the highest powers of learning, conception, and purpose in one brain, from the low-purpose stupidity and incapacity in another? In short, how can we expect to detect a disturbance of balance in the seats of mental faculties when we cannot tell whether they are there or no? These reflections lead us to expect that in some forms of insanity, and especially in acute cases or early stages, you would not find evident changes in the grey matter.

On the other hand, the term insanity may be used in a general way to include any grave mental disorder; its definition, "disturbance of self-consciousness, based upon delusions," will include the delirium of fevers, pneumonia, meningitis, or alcoholic or other intoxication, as well as the demented state of chronic mania and general paralysis. Now, in the cases of acute meningitis, fever, &c., we do not term the person insane, however mad he may be; his insanity is reduced to the relative dimensions of a symptom.

It is necessarily true that all insanity has a cause equivalent to the fever or meningitis which produces it in what we call delirium, and it is this cause which the morbid anatomist searches for. When he has found it, insanity will become a symptom of the disease he discovers.

Until then the insanity is ultimate, in the same sense as essential epilepsy or tetanus or sciatica is ultimate.

In some forms of insanity we are very far from this end; such forms as you may meet with in criminal lunatic asylums, where the delusion, perhaps, consisted in a wrong notion that the property or life of another was a trifle beside the heat of morbid passion or greediness in the grey matter of the individual, popularly called his heart. In this part of the wide scope of insanity, where madness is scarce distinguishable from badness, no doubt it will be long before the cerebral alterations that correspond are known; this will be discovered, probably, when normal mental phenomena are traced to a material basis.

But in the case of general paralysis especially, if in no other form, it is questionable whether we have not already discovered such evidences of morbid anatomical change, as will justify us in regarding that disease as a chronic general meningo-cerebritis, to which the insanity is phenomenal, but this must be still held as a question, for you will notice, that the grave changes that characterise this disease, are very like the kinds of change in the spinal cord in locomotor ataxia, and no question is more hotly contested at the present time than that which asks whether in locomotor ataxia we deal with inflammatory disease of the cord, or whether the observed changes are due rather to the consequences of congestions having their rise in functional irritation; and thus it might still be held that the morbid appearances of the cortex of the brain in general paralysis are due to functional excitements primary in the mind itself.

We will now enumerate to you the chief morbid appearances that have been found in the brain.

Acute Mania and Melancholia.—In some cases of acute mania there is no discoverable lesion after death—that is, no lesion that we can at present discover. In other cases appearances are found in the cortex of the brain allied to inflammation, and yet not amounting to a distinct inflammatory process. The disease appears as a low degree of red softening, and it is said by some, as Baillarger and Pinel, that the softening affects particular layers of the stratified grey matter. Stress has been laid upon capillary extravasations of blood, which are found chiefly in the form of effusions into the perivascular sheaths, or capillary dissecting aneurisms, as they are called; but some blood-corpuscles also burst free into the parenchyma, and are there converted into hæmatoidin pigment. Besides these extravasations, fatty changes are found in the nuclei of the capillaries, as well as irritative multiplication of the connective cells about them, while the proper nervous elements in this acute stage undergo no well-defined alterations.

Chronic Mania and Melancholia, &c.—In these chronic conditions the surface—that is, both membranes and cortex of the brain—usually show the alterations we have described as atrophic induration, the membranes being thick and the substance of the brain wasted. The minute particulars of the process are traced as consecutive to the softening in the acute stage, such softening being succeeded by induration. The effused blood is now in the form of pigment around the minute vessels, and the spread of new connective has induced hardening of the affected part. This excess of connective is accompanied by colloid and amyloid corpuscles, as usual. Meantime the nerve-fibres break up and the cells are more or less defaced, becoming pigmented, or, according to some, their nuclei multiplying, and, according to others, even calcareous degeneration occurring in them.

General Paralysis of the Insane—Dementia Paralytica.—In this disease the brain and spinal cord, together with the membranes, have undergone a degeneration by a chronic inflammatory process somewhat like that found in the kidney in Bright's disease. The disease has often a duration of five years, and then terminates fatally. After death there is found a marked degeneration or destruction of the most important parts of the brain. The grey substance of the cortex suffers more especially, but no portion of the cerebrum is excluded from the process. The visceral arachnoid and pia mater are much thickened and closely adherent to the surface, so that on an attempt to remove them the cineritious structure is torn. In this grey substance the ganglionic cells are found to have completely degenerated and altered in form and colour. Corpora amylacea are present, and a quantity of new connective tissue which binds the whole together and hardens it. The process is described by some as commencing on the surface, so that in the early stages this is soft and clings to the membranes in separation, but at a later stage the softening penetrates deeper while the superficial layers are undergoing consecutive induration. Thus, the process, marked by a stage of softening succeeded by induration, spreads from without inwards in the cortical grey matter. It is during this change that the granular nuclei and colloid corpuscles replace the nerve-cells; some think that the latter give rise to the former by degenerative changes. Sometimes on the surface of the brain old extravasations of blood are seen, and these may have become organized into membranes. In many cases most striking changes have taken place in the blood-vessels. Thus, in a specimen sent to the museum the vessels stood out like so many bristles when a section was made, and could be pulled out to the length of two or three inches, having undergone a remarkable calcareous change. Observers have also shown how the vessels undergo a morbid change by the thickening of their walls, by which they put on a varicose appearance; also Dr Sankey has pointed out that the sheath dilates in a spindle-form widening, and the vessel is convoluted within.

This state affects the smallest arteries, and has received much attention. Wedl says the veins become fibrous and obliterated. Some have thus regarded the disease as one primarily of the blood-vessels, and as due to an hypertrophy of the connective tissue in the small arteries and veins of the pia mater of the cortical structure of the brain. Cases have been recorded where the medullary matter was so hardened by the chronic inflammatory process and the production of adventitious materials that the grey substance could be scraped off, leaving the white matter beneath in the forms of the convolutions. The spinal cord is also wasted, and is found to have undergone degeneration. It is asserted by Westphal that the spinal cord in this disease shows the same grey degeneration of the hinder columns as we find in locomotor ataxia; this has not been so in several instances that we have examined; while in ordinary locomotor ataxia the functions and structure of the brain are unimpaired.

After thus enumerating the minute changes which have been described as associated with insanity, we must warn you that there is nothing peculiar in their kind. *There is no one of these changes which may not be found in ordinary brains after middle age or even younger.* The most characteristic point with regard to insanity is the universal spread of these changes. Local causes of irritation, such as tubercles and hydatids, do not of themselves produce insanity; they are rarely found associated with it. To produce insanity the morbid changes must be widely diffused in the grey matter of the surface. Most people have some bad capillaries and colloid corpuscles as well as over-pigmented nerve-cells. This may remind us that similarly most people are suspected to have a mental flaw by some very intimate friends. These morbid changes are found, as you would expect, chiefly developed in old cases, where the mind has been almost totally done away. The organ of the mind is then found wasted, but the wasting process is of a kind which leaves it doubtful whether functional change caused it or was caused by it.

Spinal Disease and Injury.—Death occurs in these cases through the instrumentality of some organ which has become impaired in function by the loss of its proper nervous influence; the consequences vary with the seat of the disease or injury. If a fracture of the spine occur in the first three cervical vertebræ, death is instantaneous; if it occur in the cervical region below the fourth, so as to leave the phrenic nerve untouched, the chest is paralysed, and respiration is continued entirely by the diaphragm; but then death takes place in a few hours. In such a case the lungs are found excessively gorged, and the blood almost starting through the tissue—indeed, blood is sometimes found mixed with frothy mucus in the bronchial tubes; as the injury occurs lower down in the dorsal region of the spine, so is the chest less extensively paralysed, and life is prolonged for a proportionally longer period; if the injury be too low to cause death very quickly from paralysis of chest, then other changes occur in the paralysed parts below, but much more slowly, and those which suffer more especially are the bladder and the back: we have already alluded to the question whether the inflammation of these parts be due immediately to the nervous influence being removed, or merely to retention of urine in the case of the bladder, and to continued pressure from immobility in the case of the back. From the bladder being paralysed, the urine is retained and becomes ammoniacal, so that the mucous membrane is inflamed; and the inflammatory condition may proceed up the uterus to the kidneys, which, thus becoming involved, suppurate, and death speedily ensues. This suppuration of the kidney is, we believe, the most frequent cause of death in all cases of injury to the spine which have not occurred high up, and also in cases of chronic disease of the spinal cord. At the same time, with the paralysis of the bladder, a bed-sore forms, and this is a cause which associated with the renal disease may lead to a fatal result; sometimes the bed-sore is sufficient to kill by itself, and we have twice seen this so extensive that, the sacrum becoming necrosed, it has set up a secondary inflammation of the membranes of the cord added to its original disease. In most cases, then, of fatal spine disease you may find the cause of death in inflammation of the urinary organs, and bed-sore; occasionally the suppuration has infected the blood, and lobular abscesses in the lungs may be met with: in three cases we have seen such extensive sloughing of the bladder that a general peritonitis was set up.

DISEASES OF THE HEART

In cases of heart disease with obstruction of the circulation we find the morbid conditions remarkably uniform. Whether the cause be a contracted mitral or aortic orifice, or whether it be simply a dilatation of the heart, you always find the same effects, provided only that the disease be uncomplicated with acute endocardial inflammation. These effects are consequent on the impediment to the blood, and their constancy makes it convenient to use such a term as *ischæmia* for such an impediment, the immediate cause of all these effects. We have already mentioned the changes in the form of the heart caused by the various valvular affections, as well as the outward appearance of the body, characterised by the dropsy of its lower part, and the slight yellow tinge of the upper. The ischæmia which leads to the exudation of *serum* in the legs, also produces some effusion in the abdomen; but this is generally not to any great extent. We would ask you to notice the extreme wasting, which, more or less concealed by the fluid, is present in cardiac dropsy; this emaciation is generally really greater than that so much more striking and generally recognised which characterises pulmonary phthisis. There may be some fluid in the chest, generally on both sides, but sometimes limited to one; the side on which it occurs is then often determined by the position of the patient during the last few days of life, or sometimes by the fact of pleuritic adhesions existing on one side, and thus necessitating the effusion on the other; or it is possible that it may be caused, though this is not yet actually proved, by the pressure of some particular part of the enlarged heart upon the root of the lung. The *lungs* themselves, from being chronically congested, undergo that peculiar induration which we have mentioned under the name of "splenization;" and if the pressure of the blood be very great, the vessels (probably the pulmonary veins) give way, and apoplexy of the lung is produced; but as at the same time the arteries are always found plugged with coagula in the apoplectic spots, the pathology of the hæmorrhage is subject to doubt; the *bronchial* membrane is highly congested, and tubes filled with mucus; the *liver* is large, and of a dark purple colour externally, and within is seen to be in the condition known as "nutmeg;" the *spleen*, like all the organs in the body, is indurated, and rarely contains fibrinous masses; the *kidneys* also are very hard, and also may contain such masses; the latter most usually occur where the diseased valves are covered with vegetations, but we cannot say that this is invariably the case. The mucous membrane of the alimentary canal is also highly congested; that of the *stomach* is of a dark red colour, covered with tenacious mucus, and the membrane sometimes abraded, or in the condition known as hæmorrhagic erosion; the *intestines* are also much congested, as well as the *uterus*. All the appearances we have named are generally found in those who have died slowly of heart disease, and they all point to an excessive congestion induced by ischæmia and engorgement leading to induration of the parenchymatous viscera, to effusions of serum from the serous membranes and of mucus from the mucous surfaces; the conditions of these membranes, together with the secretion, sometimes indicating a state which can be called by no other name than inflammatory.

There is another very different class of cases where death is due to ulceration of the valves and consequent blood-poisoning, while there is no ischæmia, or very little. There may be no external dropsy or but a trace of it, and only a small amount of fluid in the serous cavities. The surface of the body is yellowish. The viscera are soft, flabby, or even pulpy, especially the spleen, which is large, often of great size, ranging in weight from sixteen to forty ounces. There are usually serous inflammations, and embolisms of the viscera are more common than in ischæmia. Indeed, we may say that embolism properly belongs to this class of cases, and if the ulceration of the valves is severe the parts that are embolized may suppurate, so that in this way abscesses may be set up in the brain, spleen, kidneys, &c., constituting a kind of pyæmia.

Acute pericarditis is often a cause of death in Bright's disease, and as a sequel to other chronic affections; but probably, in its simplest form, is rarely or never fatal as an idiopathic affection. If rheumatism, for example, is fatal by pericarditis, it is usually on account of the pleurisy which is combined with it; or if it should cause death alone, it is generally by effusion, which embarrasses the heart, and not by the intensity of the inflammation. Judging, therefore, from our own experience, simple pericarditis is rarely fatal, except as a sequel to other disease; but if itself fatal, this is not so much from the violence of the inflammation as from the fluid effusion which results; such fluid, therefore, may generally be expected in a fatal case of rheumatic inflammation.

In *aneurism of the aorta*, very often no other disease of the body is found, except in the arteries, and that may be local and acute; but in advanced age aneurism may be associated with various degenerations of the tissues, as fatty heart, granular kidney, &c. When aneurisms press upon the air-passages they set up a very well-marked form of granular or small-lobular pneumonia, which is the common cause of death. If an aneurism burst into the air-passages it kills rather by suffocation than hæmorrhage, so that the organs may be congested. A fatal hæmorrhage into a serous sac amounts to from three and a half to seven pounds of blood. If an aneurism burst into the cellular tissue and becomes "diffused," it may be fatal with a much smaller loss of blood, shock being much greater. Some cases die of pyæmia through gangrene of the sac or as the results of operations. There are other accidents too numerous to mention.

In fatal cases of *angina pectoris* which we have seen the heart was very fatty, and the coronary arteries were ossified; one of the specimens we have already shown you.

DISEASES OF THE RESPIRATORY ORGANS

Bronchitis.—Although this is one of the commonest of English diseases, yet it is rarely fatal in its acute form, at least in otherwise healthy adults; so that if any one not very young nor very old should seem to die of acute bronchitis, acute tuberculosis, &c., should be suspected. Even when death is produced by chronic bronchitis, other morbid affections are associated with it; thus, it is very frequently attendant upon many other diseases, such as morbus Brightii, and is often then the immediate cause of death. The fatal cases of simple bronchitis are generally those where a subacute attack ensues upon an old bronchial disorder, and in such examples you very frequently find emphysema of the lungs and hypertrophy of the right side of the heart. In consequence of the mode of death, all the organs are gorged with blood and the legs may be œdematous; but although, as we have stated, long-continued congestion of the liver leads to the nutmeg condition, it is remarkable that this is rarely seen in bronchitis, or at any rate only an incipient stage of the change is found. In death by this disease you must open the tubes, and particularly examine the smaller ones, for these you may find filled with purulent mucus and perhaps dilated, &c., whereas if you are content with merely cutting across the lung you may be at a loss to account for the fatal result. But if you find the right side of the heart hypertrophied, you will, of course, be sure of some long-standing impediment to the circulation.

Pneumonia.—In by far the majority of cases where pneumonia is found, you will also discover some pre-existing and more chronic disease in some organ of the body. In primary pneumonia the lung is hepatized at the time of death, and on its surface there is generally more or less lymph; all the organs are gorged with blood, as might be supposed, and some change has probably even occurred in their secreting structure, judging both from appearances after death and from symptoms observed during life; thus, jaundice is sometimes an accompaniment of pneumonia, although after death no marked change is discoverable in the liver tissue besides engorgement; the kidneys too, which very probably have produced albuminous urine during life, are now found swollen and coarse-looking, whilst they show beneath the microscope their tubules filled with granular contents. If any urine be in the bladder, you may test it for chlorides, which are said to be in very small quantity, or altogether absent in pneumonia; this we can verify from having examined it in many cases. The best way is to make a comparison with some healthy urine, when you may be sure of the result; if you take the latter in a tube, and add some drops of nitrate of silver, a dense curdy white precipitate will occur, sufficient to fill half the tube; but if you take some of the pneumonic urine and test it in the same way, first adding a little nitric acid to dissolve the phosphates and other salts of silver, you will produce in it only cloudiness. It is not yet proved that such an absence of chloride of sodium does not also accompany other febrile disorders; but at present, we believe, the theory is that all this salt is exhausted in the rapid cell formation which takes place in the lung, and thus the blood, and consequently the urine, are deprived of it.

After seeing a number of cases of pneumonia you will notice that the extent of inflammatory change present in them severally is subject to great variety, so that you are obliged to seek some explanation of the fatal result in those that show less extensive disease; for remember it is not enough that you should find "pneumonia" and say the person died of it; you should then learn *why that particular patient succumbed to the disease*, an inquiry too often forgotten or slurred over. Sometimes the intensity of the pneumonia is evidently very great, there is purulent infiltration or even gangrene of the lung; but sometimes neither the extent nor the severity of the morbid change suffices to make clear the cause of the patient's death. You should then examine the alimentary canal for that catarrhal condition which we have before spoken of, also notice whether jaundice exists. Further, you should make a general survey as to the integrity of the great viscera, which may be found evidently diseased. In the young, acute pneumonia is, you know, less fatal than in those more advanced in life; this would point out that the deterioration of the great organs withdraws from the lung such aid as they can afford to it when they are fresh and vigorous. You remember the herpetic lip so common in pneumonia; this clearly indicates the generalised attack which the patient is undergoing, and, with other facts to which we have alluded, proves that acute pneumonia is a disease of more than the lung.

If you find a pneumonia in the form of small isolated patches, in other words "lobular," remember that this is always secondary to some other disease, which you must discover—pyæmia, some disorder of the upper air-passages, fevers, &c.

Phthisis.—We fear that the effect of recent discussions has not been to make clear the nature of this disorder, nor even to decide the scope and meaning of its dreaded name. The prevailing disposition amongst pathologists is to consider phthisis as comprising several entirely distinct disorders; this is nothing new, but is rather a reaction from the relatively more modern view of Laennec and Louis, who regarded phthisis as the result of tuberculous disease of the lung, and a reassertion of the views of Bayle and other older writers, who held phthisis to be "granulous," "ulcerous," "tuberculous," &c., making the tubercles characterise a particular kind of phthisis, instead of declaring them to be universal in and, indeed, the sole cause of phthisis. We will not enter again here upon this question, but only make a few remarks, which may tend to guide your expectations as to the circumstances of the fatal issue in different varieties of phthisical cases.

You will find the examples of phthisis, which come down, almost monotonously alike in certain main features; but if you study in detail their varying characters you will find practically unlimited differences, indeed each case will offer peculiarities of its own. Now, the main and constant characters are those which mark the anatomical appearance of the lung; they are—*Firstly*, the disease is destructive of the tissue at the parts affected; *secondly*, it attacks the lung from above downwards; *thirdly*, the diseased parts put on a more or less rounded form, and are firm to the touch, so that when small they feel like knots or grains in the texture, and can be distinguished by one's finger, without the aid of sight, from any other disease of the lung.

These general features are so constant, that we regard them as sufficient to show that phthisis in all its varieties is essentially one and the same disease.

The characters that vary are those that more concern the symptoms and termination of the disease. Thus, you will find that while the phthisis spreads from above downwards, and destroys the part it involves, and produces hardish knots in the tissue, features common to all phthisis, there will be additional appearances according to the stage of the disease and the mode of death. The modes or causes of death in phthisis, we should say, may be considered as follows:

Firstly.—Hæmorrhage, acute pleurisy, pneumothorax, acute pneumonia, miliary tubercle, repeated tuberculo-pneumonic destruction of the lung with pyrexia—any of these may bring the patient down unexpectedly, so that, instead of the extreme emaciation that we are accustomed to look for in phthisis, the body may be comparatively well nourished; this is especially the case with hæmorrhage, pneumothorax, or general tuberculosis, so that if the subject is well nourished either of these accidents may be anticipated.

Secondly.—If the case has passed lingeringly on through a longer period of illness, and the emaciation is greater, embolism of the arteries of the healthier part of the lung is a frequent cause of dissolution, or diarrhœa from extreme intestinal ulceration (we have twice met with perforation from tuberculous ulcer of the ileum, and once with fatal constriction from contraction of such an ulcer, but these accidents are rare). In such more prolonged cases destruction of the lung is more complete and there is greater pigmentation, and induration of the organ.

Lastly.—If the disease has been protracted for years, then other appearances are presented, and fresh consequences declare themselves. The disordered part of the lung is contracted by the cicatricial action, which was indeed necessary to the prolongation of life; and it may be that the remainder of the lung is expanded to fill the thoracic cavity, constituting pulmonary emphysema, and inducing bronchitis, dilatation of the right heart, and perhaps dropsy, and completely transforming the characters of the malady as well as its mode of termination; so that if met with at first in its latter stages, the complaint may not be recognised as phthisis at all, especially as the tubercles, which individually never last long, are not now visible, and may be with difficulty traceable in most parts of the organ. Some special writers on the subject of phthisis call this a distinct kind, and, without showing any adequate reason why, refuse to recognise that the protracted course of such a case necessarily infers extensive cicatricial action whose product is necessarily fibrous or "fibroid." In these protracted or "fibroid" cases the lardaceous change of the viscera is common, and therewith you get albuminuria, and the case may thus put on the characters and mode of termination of Bright's disease.

Thus, excluding cancer, the lung in phthisis will offer a compendium of all the pathological accidents to which that organ is liable; and differences thus arise enough to confuse those who seek for variety, while meantime the leading features of the disease are identical in all cases, as we before have said. The study of the various accidents in phthisis will, we hope, prove of the utmost interest to you.

DISEASES OF THE ABDOMINAL ORGANS

In cases of death by abdominal disease you will be struck with the sunken aspect of the visage; the fleshy parts of the face shrinking and the outlines of the skull coming into view, so causing a ghastly aspect which is not observed in the faces of those dead of thoracic or cerebral ailments, &c. This remark, however, applies rather to diseases of the alimentary canal and peritoneum than to those of the parenchymatous abdominal viscera. During life the same expression of countenance is generally to be observed in these cases, and it is sometimes alluded to as the "abdominal face," which is never disregarded by the experienced clinical observer.

Notice always any alteration of natural proportions in the figure of the abdomen, any elevation of the lower right ribs indicating a tumour or abscess of the liver, &c.; we have occasionally observed that cases of hepatic disease have become much more certain in their nature at post-mortem inspection, even before any incision was yet made in the body, because a bulging of the right lower ribs was plainly disclosed when the body was seen laid entirely bare. You should remark that the liver may be very large through congestion, fatness, and other equable swellings, without bulging out the ribs; while a much smaller increase of the liver through the presence of a tumour or hydatid will surely lift the ribs.

Before the body is opened the abdominal viscera should be examined again carefully; if death is already of some hours' duration, the gases accumulating in the alimentary canal probably will have made palpation difficult, so that it is well to make the examination as early as possible. The impression made upon you when the opinion you have formed is immediately verified or belied by a disclosure of the secret you were endeavouring to discover is lasting beyond all comparison with knowledge gained from more doubtful and imaginative sources. The experience of a single afternoon will thus remain indelibly stamped, when whole months of reading have left no trustworthy remainder.

One great danger of disease of the abdominal viscera arises from the presence of the peritoneum, which may become acutely or chronically inflamed; or the whole membrane may be involved in tuberculous or cancerous action, or either hepatic or other obstruction may cause serum to collect, causing ascites.

In old cases of disease of the bowels, and in all cases of ileus, it is necessary in the progress of the examination to be careful to note well the state of the parts as you turn any into new positions; also to observe what force you use, since you are likely to find perforations of the intestine, and you ought to be able to be quite sure whether it was possible that you might have caused the rupture of an unsound spot in the bowel by your previous handling. However, you should remember, that when the intestine is perforated *ante mortem* there will nearly always be gas in the peritoneum, and a fæcal contamination of the peritoneal contents; but in very old cases of dysenteric ulceration, when the patient has lain for days in a dying state, you may find the bowel extensively perforated, without the intestine having had force enough to expel gas or fæces into the peritoneum. Likewise in bladder cases you must note the state of the bladder before much disturbing the parts; it is most unsatisfactory when a hole in an inflamed bladder is found after the removal of the intestine, and a question is raised whether the bladder may have been injured in the removal of the bowel.

We would also strongly recommend you to remove the liver, stomach, and duodenum, and the parts around the bile-duct and portal vein, in their natural connection, with as little disturbance as possible, for if the liver be removed separately, important changes in the course of the bile-ducts may be irremediably spoilt.

As to the generalisations of abdominal diseases these are not usually important in the same degree as the generalisations of thoracic disease, for the latter, by the *dysæmia* and *ischæmia* they induce, give rise to more or less universal changes. There are some general disorders which have their local tangible seat in the abdomen, such as leukæmia in the spleen, Addison's disease in the supra-renal bodies, and Bright's disease in the kidney; we have already described these, but would again warn you of the necessity of inspecting carefully the other viscera in Bright's disease, since apoplexy, serous inflammations, &c., are very constant accompaniments of that disease on the post-mortem table.

But otherwise the complications of abdominal diseases are generally not many, and yet you must not fail to notice that it is quite a common thing in cirrhosis of the liver, cancer of the stomach, &c., indeed in any chronic abdominal disorder, to find moderate or slight recent pleurisy, or hydrothorax, &c., evidently produced in the last few days of life. We have already endeavoured to explain such inflammatory changes, by reference to the probably irritating influence of stagnating and impure blood in dependent parts.

GENERAL DISEASES

Pyæmia.—The cause of death after great wounds or extensive sores, or gangrene, was formerly little inquired into; it was ascribed to shock, fever, or such like vague considerations. But when post-mortem examinations were made, it was found that internal inflammations are present under these circumstances, and often are evidently sufficient to produce a fatal result. These inflammations, however, are of a peculiar kind. Their most marked feature is a tendency to the rapid formation of pus; hence it was natural to think that the pus had been taken up from the wound or sore, and lodged in the internal parts where it was found; and thus the earliest and simplest notion of pyæmia arose. But it soon became evident that the pus in the internal parts is formed by inflammation, and is not simply conveyed there; for the inflammatory process is evident around and in the puriform collection.

The idea and word *metastasis*, or transferrence of a diseased process to a new seat, here lay ready for employment; and, indeed, this vague term still serves a useful purpose in expressing the shifting of a diseased action from one place to another; still, such vague ideas, if accepted as sufficient, repress inquiry, and we must, here as elsewhere, take the facts of the disease, and the results of experimental inquiry, to throw light on its actual nature.

Such, indeed, has been the course pursued, and now we are in this position—that we have two distinct departments of facts bearing on the question of pyæmia: the facts from human morbid anatomy, and those from experiments on animals. To take the latter first, many distinguished observers have studied the effects of injecting putrid matter into the veins or the cellular tissue of dogs and other creatures; the result is the literature of *septicæmia;* but the bearing of this on our knowledge of pyæmia is not as yet very direct, for by such experiments you do not generally produce those suppurations that are characteristic of pyæmia as met with in the post-mortem room. The animals experimented on recover either with or without grave symptoms; or die, generally with diarrhœa and collapse, when the alimentary canal shows on inspection signs of inflammation after death, and there are congestions, ecchymoses, or inflammations in the lungs and other organs, that is, such signs as prove a poisoned state of the blood.

It has been said by Dr Burdon-Sanderson that the poison of pyæmia increases in intensity of action in animals successively inoculated with it one from the other; so that one animal, inoculated with matter from the peritoneum of a former victim just dead of disease caused by inoculation with bacteriferous septic matter, will suffer more violently and die quicker than the first victim. And a third animal, inoculated from the second, will die yet quicker. So that it appeared to Dr Sanderson that the poison was capable of intensification by a sort of cultivation. These experiments were repeated by Dr Goodhart acting with us, but we have, so far, not been able to confirm those results. A first dog died when bacteriferous fluid was injected into the peritoneum, but the second dog, when injected with fluid from the first dog's peritoneum, survived with but slight symptoms.

We have, with Dr Goodhart, examined the blood in many living cases of pyæmia, grave erysipelas, &c., without finding any bacteria in it. In making such an observation one must remember that the blood is apt to contain minute particles, similar to the fat-molecules of chyle; and it requires a microscope of excellent defining power to observe these, and distinguish them from bacteria. Such particles you may not unfrequently find in the blood of dyspeptic persons; we have found them in some excess in typhus and other fevers, but never in such quantity as to draw a marked distinction from dyspeptic cases. We have not as yet succeeded in discovering any bacteria in the blood of living pyæmic cases; they are often seen in the discharges of open wounds; we have not yet found them in matter from a hitherto unopened abscess in a living person.

They quickly appear after death, and in post-mortem examinations, made a few hours after the patients' decease, we have found enormous quantities of bacteria, both in the products of inflammation, and to a less degree in the blood. We have seen appearances that would indicate, but not quite certainly, that the bacteria arise from disintegration of white blood-cells or of pus-cells. What part these bacteria play in the causation of septicæmia is as yet uncertain, but there is no inquiry which is of greater interest than this question, nor any that promises more valuable and scientific and practical results.

In our experience of deaths from wounds, sores, &c., we have not met with any class of cases that at all closely correspond to these results of experiment by showing enteric disorder, collapse and quasi-choleraic phenomena. So that we think it is better at present to regard the history of the experiments on animals, that we have alluded to, as a distinct branch of the history of septic infection, whose bearing on clinical pyæmia is as yet a problem requiring solution.

But if we take the morbid appearances in the human body after death from grave wounds and sores, these form a very definite series, one to which the term *pyæmia* properly applies. We have already described the appearance of the changes in each individual organ, and will now only consider their distribution in the body and their general features so far as will serve to give us a view of the nature of the disease. The most frequent seat of the abscesses is in the lungs, and as these organs receive the blood from all parts of the body, this at first favours the idea of a simple transfer of the pus or of some cause of pus, in the blood, from the sore to the lungs. In other cases the liver is especially affected, and it is interesting to notice that in these cases the sore is generally in the alimentary canal, from which the liver draws its blood. These two facts raise a very strong presumption that the cause of the pyæmic suppuration in the lung and liver is the introduction of pus, or of a pyogenic something, into these organs from the sore. The proof of this is rendered absolute *in some cases* by our finding, in the pulmonary artery in the lung, or in the portal vein in the liver, a portion of purulent clot, while the veins of the sore contain similar clot; and in all cases the peculiarly strict circumscription of the pyæmic inflammatory affection in the lung or liver corresponds suggestively with the like limitation of the distribution of individual branches of blood-vessels in the parenchyma of the organs, thus indicating a dependency of the local disease on some occurrence proper to the vessel.

In some cases no doubt can exist that the lung or liver is thrown into inflammation by the advent of a purulent embolus actually found in the vessels, and thus we have a sufficient explanation of the suppurations in those cases of pyæmia. They arise from embolism, and some would therefore speak of *embolic pyæmia*. We will consider the propriety of this presently. Others, especially in Germany, go so far as to hold that all secondary abscesses arise thus from embolism, and so make all pyæmic suppuration a department of embolism. This we shall see at least the following reasons to think is not correct.

Thus, if we observe the *position* of the pyæmic abscesses in the lung, we find that a very great part of them are limited to the lower lobe, and to the dependent part of the upper lobe—if any at all are present in it; while the ascendant part of the upper lobe is generally quite free, and when affected has far fewer than the lower one; for example, in one instance out of many the lower lobes contained thirty-three abscesses, the upper only three. Now, if the suppuration depended *only* on the casting of emboli into the pulmonary vessels, it would be more likely that the upper lobes would suffer; because when a person is lying in bed the breathing, and consequently the circulation in the lung, is freer in the upper part, so that thither the emboli would mostly be carried. Again, although the lung is more generally affected in pyæmia than other organs, yet sometimes you find pyæmic abscesses in the liver, &c., as after injuries to the head, when there are no abscesses in the lung. Yet any pyogenic matter must get through the lung on its way from the head to the liver, and one knows no mechanical reason why matters which have passed the pulmonary should stick in the hepatic capillaries. Again, it is yet more frequent to meet with cases, generally more chronic in their course, where the abscesses all form in the external parts of the body, that is, in the teguments, in the muscular tissue, joints, &c. Such cases may recover through the freedom of the viscera from implication. The affection of the joints is often so marked as to justify the term *rheumatoid pyæmia* as applicable to these; and when we consider that through the medium of gonorrhœal rheumatism and scarlatinal rheumatism these cases are linked to rheumatic forms of arthritis, we find ourselves getting a long way off the embolic pathology we have just described, *i.e.* because the regions attacked are not such as are exposed to embolism; and when, again, we know that cases of pyæmia occur entirely without any kind of wound or sore—we have ourselves seen several cases of idiopathic pyæmia where the most careful search showed the absence of any primary sore—then we find ourselves in another way deprived of the embolic theory, *i.e.* because there is no showing any source for the emboli. Lastly, we must remember that some kinds of pyæmia appear to elect proper seats of secondary suppuration, especially the pyæmia which follows suppurative periostitis, and which produces abscesses in the heart and kidneys, usually without affection of the liver; while, on the other hand, injuries of the cranium, affecting its sinuses, often produce abscess of the liver. Again, pyæmia after typhoid tends to affect the salivary glands; that after scarlatina to affect the joints.

The above facts, taken severally, show that embolism is not the only cause of pyæmic suppuration; taken together, they go far to reduce embolism to the dimensions of one incident, which, among others, determines the localisation of pyæmia on special points.

Now, nothing can be more satisfactory than the history of embolism; we can so well understand how a clot from a suppurating part may be moved on or from that part, to set up the same suppuration elsewhere, that the clearness of our knowledge of the incident wants nothing. When, however, we take the rheumatoid cases, which affect chiefly the joints, or other numerous cases where, without discoverable emboli, the lower lobes of the lung are chiefly affected, the explanation is not so clear and suffers by the contrast.

Many facts go to show that an impure state of the blood produces greater effects where there is stagnation of it. This is plainly indicated by the position of the abscesses in the dependent part of the lung, but the same truth is also shown by the suppuration of the veins in which thrombi have formed. This we have seen in several marked instances, especially about the prostatic plexus, or behind the valves of the great veins, or in the cranial sinuses. Here the explanation is almost as clear as that of embolism, for it is certain that when the blood is impure the impurity will be increased by accumulation where it ceases to circulate, since the effect of circulation is to bring the blood under the action of purifying organs. This we see is so certain that the explanation of the occurrence of suppuration in dependent and stagnant situations is almost as secure as that of embolism; and it is quite possible that the two conditions may coexist, so that when small emboli are sown broadcast in the vessels of the lungs, those which lodge where the blood is purified freely in the upper part of the lung may not be able to excite suppuration, while those which stay in the stagnant lower lobes, already œdematous and scarce breathing at all, suffice to arouse the inflammatory process.

From these considerations it would seem that if we may use the term embolic pyæmia as signifying cases where embolism is proved to occur, then we may also use the term *stagnative pyæmia* to indicate the formation of abscesses by the stagnation of poisonous matter within coagula in the affected part. The other cases we have alluded to, where the joints, kidney, or liver, are especially selected, are not at present susceptible of more than such hypothetic suggestions as that the poison has a *natural affinity* for the parts that are inflamed. For when we neither have the embolus nor stagnation to explain the local affection, as in cases of the special disorder of the joints in rheumatoid pyæmia, the glands after fevers, the kidney and heart after suppurative periostitis, then we are obliged to assume an attraction between the part affected and the pyogenic matters in the blood. Of course, this is unsatisfactory when compared with the clearness with which we can follow the embolus in its course or anticipate the effects of the stagnation. But it is the best that we can do at present, to consider the fixation of pyæmia in the joints as analogous to the fixation of smallpox in the skin and of typhoid in the intestine.

After this general survey of the indications of its morbid anatomy, we must regard pyæmia as made up of blood disease and local inflammations. We can trace several ways in which the blood disease and local inflammations are related to each other.

1stly. The general disease may greatly predominate, so that the blood becomes unviable, and death occurs with signs of blood poisoning, revealed during life in the form of high fever and rapid fatal exhaustion, and after death by softening of the great viscera and extravasation of blood and ecchymoses within the tissues, no local suppuration being found. Also here we must remember that some cases of pyæmia of a most characteristic kind are found without any kind of sore whatever; we should not say this so strongly unless we had in several instances searched with the utmost care through all parts of the body and found no primary disease.

But in many cases such pyæmia as this is traceable to th of ordinary fevers, such as scarlatina and diphtheria. It is especially to the pyæmia of the puerperal state that this applies; the results of Dr Hicks' observations showed that in by far the majority of the cases of puerperal fever the patients had been exposed to contagion of some known fever. The existence of a proper pyæmic contagion is a matter much contested at present, and there is a farther contest among those who would admit such a contagion upon the question whether the poison enters by the wound or by the natural surfaces of the patient. Such facts as Dr Hicks has adduced would show that contagion, in some cases at least, is introduced as in other fevers, while the at least partial success of Lister's antiseptic plan of treating wounds would suggest that the wound itself is a principal entrance for some pyæmic contagions. It will naturally be thought that the pyæmia arising from scarlatinal poison is not properly pyæmia at all, but in the present state of the inquiry Dr Hicks' evidence would show that, if it is not so, such fever is at present confounded with pyæmia; in other words, that clinical pyæmia *now* includes many cases of various forms of fever, modified by the products of a suppurating placental or other sore.

2ndly. In another kind of pyæmia the wound or sore produces a morbid matter, which is absorbed and so poisons the blood. It is probable that the pyrexia of surgical fever is principally caused by the absorption of the products of the wound, so that this kind of pyæmia is allied to common surgical fever. The prevention of putrefactive changes in the discharges of the wound averts the danger of pyæmia from this cause, and the success of the antiseptic method shows sufficiently the importance of this source of blood poisoning. It, however, creates only one kind of pyæmia, characterised by it appearing later and running a more chronic course, with secondary suppurations. It is believed by some that there is a pyæmic *contagium*, which acts first upon the wound and enters the system there, but further observations are required to substantiate this hypothesis.

3rdly. In some cases that go by the general name pyæmia the whole affection is confined to the formation of purulent thrombi in the veins, and the conveyance of these to the lungs as emboli, where they set up secondary suppuration. Such a process is too simple and clear to need further remark.

4thly. In these three divisions, each of which is represented by some unquestionable case, we have three *kinds* of pyæmia, kinds, that is, in the sense that they are disorders of an entirely distinct nature, but we would wish you to notice that they are not different kinds in the sense of being incompatible or necessarily alternative. Far otherwise, they may all coexist; it is quite possible that a wounded person may breathe the contagion of a fever, or some other foul matter, his blood thus becoming poisoned; and that as a consequence of this, the secretions of his wound may be more putrescible, thus producing noxious matter that is absorbed in the blood, adding another depraving element, whose tendency is to set up remotely the condition present in the wound; and, again, that the low state to which nutrition is reduced may render the veins vulnerable, so that these inflame and produce purulent thrombosis, from which embolism conveys portions to start abscesses elsewhere. And although we do not prefer the lumping together of distinct alternatives into one view, yet it is probable that, in the genesis of pyæmia, this association of distinct elements in its causation actually does occur; and that pyæmia is first started by a depraved state of the blood, and that this reacts on the wound, causing in itself and in the vessels around the putrefactive and phlebitic changes, which lead to these internal suppurations, which give to pyæmia its distinctive characters and its name.

The view we have given, then, may be thus summed up:—Simple embolism or purulent absorption or general fever, due to contagion, severally form distinct varieties of what is called pyæmia; but in many cases, perhaps the majority, all these conditions exist together.

Puerperal Fever.—We have already described the inflammatory processes in the uterus and its appendages which may follow parturition; and, when speaking of these, remarked that in proportion as fever following parturition was ascribable to simple inflammation about the uterus, in such proportion is it unnecessary to suppose the existence of any specific puerperal fever. But in a considerable division of such fevers the local inflammation was trifling or absent, and the disease comes under the pathology of pyæmia. When speaking just now of pyæmia, we mentioned Dr Hicks' observations on puerperal fever, those in which he showed the frequency of contagion from ordinary fevers; indeed, it appears, that any general distinction between puerperal fever and pyæmia, rests only in the relative frequency of contamination by fever poisons, which is much greater in the puerperal cases.

For the rest, inspections of bodies dead of puerperal fever will offer you examples of all the varieties of pyæmic accident we have before described.

Typhus Fever.—The characters of typhus during life are those of a simple fever, generally without complication; and after death we generally find no local anatomical disease. And yet the conditions of the blood and viscera give evidence of great changes, so that although there is *no definite morbid anatomy* to typhus, yet we think that an experienced observer would be able to refer the death to this disease by post-mortem examination; unless decomposition had already set in, when this would be impossible, because the changes in the blood and viscera in typhus are almost identical with those brought about by incipient putrefaction. Decomposition in typhus commences early.

The petechial part of the mulberry rash persists in the skin, and on examining the viscera these are found to be soft and full of dark fluid blood. The heart is flabby and lacerable, its fibres often granular under the microscope; this condition of the heart has been described as a special disease in fever by Louis and others; it is found in most cases, but is not absolutely constant; the heart may appear healthy and well contracted, and sometimes Zenker's waxy degeneration has been found in it.

The brain is healthy in typhus, though it has been asserted that the cerebral capillaries contain clumps of white corpuscles to account for the delirium, &c., in fever; this, however, we cannot ourselves support, the appearances figured being such as we have seen in ordinary conditions. It is well to remember Dr Murchison's suggestion, that epidemic cerebro-spinal meningitis is typhus complicated with inflammation of the brain; still, at present this is only a surmise, and if now meningitis were found in any case of typhus, that case would be regarded as one of "cerebro-spinal meningitis." The lungs frequently show bronchitis, and they are congested with dark blood at their dependent parts. Occasionally you find pneumonia, generally in the lobular form, in the lower lobes. Lobar pneumonia is sometimes found, but much more rarely than in typhoid. The alimentary canal may contain blood, from purpuric extravasation; the walls are generally quite healthy, but the solitary and agminate glands may be a little enlarged, and the latter may present that dotted appearance which has been compared to a shaven beard. Occasionally dysenteric or diphtheritic inflammation complicates typhus, but this is exceptional. There may be gangrenous stomatitis or parotid suppuration in some cases. The liver is soft and the section shows a dull clay-like lustre, which is almost characteristic. The spleen is large, and very soft, its pulp easily washed away. The blood is described as containing an excess of white corpuscles, but you must not expect this to be very evident. The kidneys are large and coarse, and the epithelium cloudy with granules, or disintegrating. Dr Murchison has pointed out that after death by convulsions in typhus the kidneys are exceedingly large, and the urine albuminous; he has shown that, at the same time, urea is present in the ventricular fluid of the brain; urea may be found there, however, when no convulsions have occurred. Thrombosis of the femoral veins, with white leg, may occur, but it is not so frequent as in typhoid; it generally affects the left leg; fatal embolism of the pulmonary artery may result from it. In typhus, too, the arteries may become obstructed, sometimes evidently through embolism with ante-mortem clots from the heart; at other times no such clots can be found in the heart, and then some believe that the clots form in the arteries, a view which may occasionally apply, but we suggest, as more probable, the supposition that such clots had formed in the heart and were thrown on into the vessel while yet recent. The extremities, nose, ears, &c., may be gangrenous; it is not yet settled whether this gangrene arises through obstruction of the arteries. Grave bed-sores may arise and extend enormously, causing necrosis of the trochanters or sacrum. The bladder may inflame, through neglect of its over-distension or through careless use of the catheter.

Typhoid or Enteric Fever.—We have already fully described the changes in the intestines which characterise enteric fever; and the dangers of peritonitis, perforation or hæmorrhages, which accompany these changes. As we then remarked, these intestinal ulcerations are occasionally accompanied by so little fever that fatal perforation occurs while people are at their work; but generally the patient undergoes the "typhoid state," and the organs of the body are found presenting the gorged and softened condition we described under typhus. It is remarkable, however, that the brain is usually firm, even firmer than natural. Otherwise there is usually nothing to distinguish the general state of the viscera from that found in typhus. The heart is here, too, soft, and its fibres rather granular; or you may meet with Zenker's change, a condition in which portions of the heart are found too solid; the fibres there are easily torn, while the cut surface glistens; the muscular fibres present microscopically a peculiar waxy change already described. (See Muscle.)

We have sometimes found the larynx ulcerated, but not oftener than in other diseases involving great exhaustion, although some have thought that the larynx shows special disease in typhoid; however, a deep ulcer is sometimes met with at the root of the vocal cords. The air-passages are very apt to be inflamed, and often importantly so. The inflammation may extend into the lung, producing fatal pneumonia; this then is found in the form of lobular patches, in the dependent parts of the lungs. These patches are around the ends of the bronchial tubes, thus indicating that the source of the irritation to the lung comes down the bronchus; being either accumulated secretions, or, perhaps, sometimes matters inadvertently swallowed through careless feeding of the patient while in a state of semi-coma. The form of pneumonia here alluded to is very frequent in fevers, and often is the immediate cause of death; it is sometimes called *hypostatic pneumonia*, and is spoken of as though it arose by simple stagnation. The expectation then produced upon the inexperienced learner is that the pneumonia implicates the whole of the most dependent parts of the lungs, arising through gravitation of the blood; but we have never seen the pneumonic change so distributed. You occasionally, but rarely, meet with a large tract of hepatization of the lung, as in so-called lobar pneumonia; and sometimes there are pyæmic abscesses, or even gangrenous patches in the lungs.

You may find stomatitis, follicular or ulcerative, also suppuration of the salivary glands. The liver has the same character as we have described under typhus; it is soft, and its section has a dull appearance, like cut yellow clay; the microscope shows the cells granular and indistinct in outline. Some consider these appearances to prove the presence of early hepatitis, but there is no lymph on the surface of the liver to support this view. It is remarkable that the extensive intestinal ulcerations of typhoid very rarely lead to abscess of the liver. The gall-bladder, however, is sometimes inflamed, and has even been found perforated; we ourselves have never seen this. The spleen is large and may be dark, and tolerably firm in the early stages, as felt during life; but at post-mortem it nearly always appears soft, and full of pulp. It is loaded with blood, and there is formation of an excess of splenic elements; such soft spleens are said to burst sometimes. A pyæmic abscess is occasionally, but rarely, found in the spleen. The voluntary muscles, especially the abdominal rectus, diaphragm, adductors of the thigh, and the tongue, sometimes show the change discovered by Zenker. These muscles may be ruptured, either in consequence of that change, or without it through violent exertions in delirium when the patient is carelessly watched. Grave bed-sores or gangrene may occur as in typhus; obstruction of the femoral vein, and consequent white leg, is more frequent than in that disease. The eruption in typhoid fever disappears after death.

Relapsing Fever.—The conditions observed in relapsing fever, with the exception of frequent presence of jaundice in this disease, do not differ from those seen in typhus. This fever is rarely fatal.

Intermittent and Remittent Fevers.—Besides congestions and softening of the organs in acute, and anæmia and œdema or more general dropsy in chronic stages, there are other more characteristic lesions associated with these fevers; they go by the general name melanæmia. The blood is found to contain particles of pigment, and, as the consequence of this, the pale parts of the body may be of a dusky tinge, but the spleen, liver, and brain, suffer especially. In these organs the pigment is found within and about the vessels, as though it had lodged in and obstructed them, leading sometimes to minute extravasations of blood; for particulars of the appearance, we must refer you to the melanic state of the several organs.

Yellow Fever has never come under our observation. From the descriptions of authors, it appears there is nothing characteristic, except the jaundice and the hæmorrhages; but the œsophagus and stomach may present considerable erosions and ecchymoses.

Glanders and Farcy are names given to two varieties of *Equinia;* the virus producing these varieties being identical, but in glanders it develops its effects chiefly on the nasal mucous membrane, while in farcy the skin and subcutaneous tissues suffer most. In the nasal mucous membrane you find a frightful state; a large quantity of fœtid tenacious mucus filling and running from the cavities of the nose, while the walls of the nares show ulcers, perhaps penetrating and destroying the bones; the earlier stages of the ulcers being in the form of tubercles or knots in the congested and swollen mucous membrane. If the case be acute, the face is much swollen, the appearance resembling erysipelas, and, as you will see in these models, bullæ may appear on the skin.

But the affection of the skin usually puts on the appearance of pustules, furuncles, and carbuncles; the early stages of these are in the form of small red patches, which swell, and soften as they swell, until little abscesses are found raising the cuticle; they do not, however, arise as collections between the true skin and cuticle, but by infiltration and softening of the true skin itself. The process shows itself, microscopically, as a formation of small delicate cells in the areola of the cutis; these increase in numbers, and enlarge, until the whole affected spot appears made up of such cells, which are compared by Virchow to the tissue of granulations, so that he includes glanders in the class of tumours which he calls "*granuloma.*" In the further progress of the abscesses blood often becomes mixed with their contents; they open, and form ill-conditioned sores, under blackish crusts. The lymphatic glands are apt to be affected; chains of glands may enlarge, forming the so-called "farcy buds," as in horses. The cause of death may be, in chronic cases, a slow exhaustion by the discharging sores; the disease appearing in the form of slow chronic ulcers, which may escape diagnosis. But in the acute form the interior of the body shows the condition of a very severe pyæmia; we have found the muscles of the extremities stuffed, if we may so speak, with abscesses of a size from peas to chestnuts, while the lungs were so full of similar abscesses that nearly half the whole tissue was taken up by them. The kidneys, heart, liver, and joints, were similarly affected. During life albuminuria is frequently present.

Cholera.—We have examined many bodies of those who have died of cholera; the following were the appearances :—As a rule, the viscera showed no signs of organic disease, although repeatedly we found old long-standing morbid conditions, such as ulcerated intestine, degenerated kidneys, chronic endocarditis, &c.; these, however, were exceptional. The majority of persons attacked were healthy. In rapidly fatal cases, dying in a state of collapse, the temperature often rises after death, and the muscles may twitch for a time; the skin is livid, especially in the dependent parts, and the face has a ghastly sunken appearance; rigor mortis is generally unusually strong. The brain is natural, or shrunken a little, having then an excess of subarachnoid liquid. The lungs are in a peculiar state; they are of small size, and have a collapsed look, being dark and flabby; they are generally very light in weight. Thus, in one rapid case, that of a strong man, we found the weight to be but twelve ounces for the right lung and eleven ounces for the left. On section their texture is dry, the great vessels containing a moderate quantity of dark liquid blood. To understand their shrunken collapsed state, you must open the bronchia, when you will find them curiously dry within; now, remembering that what prevents collapse of lungs under ordinary circumstances is the quantity of the mucus in the tubes, which prevents the exit of air, you will see how the empty and dry tubes in cholera will permit of the lungs collapsing according to their natural tendency. The blood in the great vessels is dark and thick, from the loss of water. We saw no exudations on the pleura. We have never observed the pulmonary artery unusually full of blood; in one or two instances we have seen embolic patches in the lung; the heart is generally contracted closely and firmly, and consequently its cavities, especially those on the left side, are closed and empty; on the thinner right side, the closure is less complete. There is an accumulation of blood in the great veins; the blood in the heart often contains white clots, or it is gelatinous, or tarry, dark, and thick.

The gall-bladder is distended with bile, and the liver itself appears healthy; but is flabby, and rather dry on section.

The spleen is flabby, perhaps rather tough; sometimes, however, it is soft; it is notably dry on section.

The appearance of the alimentary canal is peculiar, and almost characteristic of the disease. The intestines lie in a very small space, and they are almost always quite devoid of gas, instead of, as is usual, being more or less distended with it; hence they have no disposition to uncoil or separate themselves, but form a flabby compact mass, feeling doughy, and flabby, and pliable to the touch. They have a peculiar rosy pink colour, because, the foul gases being absent, the usual staining of their blood with sulphuretted hydrogen does not occur.

Their surface exudes a tenacious mucoid secretion, not generally described, and this may be owing to its not being always present; it is not, however, uncommon, as may be best observed by separating two coils of the bowel, when a viscid mucoid string is drawn out between them. The secretion is hardly like that of recent inflammation, as the exudation is more like mucus, and is more sticky than the greasy sero-albuminous fluid of ordinary inflammation. The internal mucous surface of the intestines has a sodden appearance;

a white creamy secretion covers it, which, when mixed with a large amount of fluid, constitutes the rice-water evacuations.

The amount of fluid is variable in different cases; there are generally no natural fæces present, and there is usually no tint of bile in the contents.

The enlargement of the intestinal glands is often very remarkable; affecting sometimes, but not always, Brunner's glands in the duodenum, generally Peyer's glands in the ileum, and most constantly or universally the solitary glands both of small and large intestine. The two latter kinds of glands are sometimes so large that they give a resemblance to the early stages of typhoid fever. We might here remind you that the condition of intestine described is not much unlike that found in cases of severe poisoning by large doses of arsenic, a fact important to remember when the symptoms of the latter during life also resemble those of cholera. The kidneys are generally soft, and of a bright red colour, weighing often rather under their usual weight; sometimes, however, they are dark and congested. The urinary bladder is contracted and empty.

In the bodies of those who have died during the consecutive fever the characteristic appearances of the collapsed stage just mentioned no longer exist. Gas is now found in the intestines, and hence the peculiar colour and feel we have spoken of are gone, and the interior is found to contain bilious matter, though the glands may still be seen large. The kidneys are generally diseased, and in some cases have presented very much the appearance found in those who have died of acute scarlatinal dropsy; the organs are large and congested, weighing sometimes fifteen or sixteen ounces; the microscope shows the tubules gorged with a granular secreted matter, and very probably their pathological condition is similar to that of the scarlatinal kidney, as the symptoms of the two during life also accord. We have seen some cases of cystitis and suppuration of the kidney in the secondary fever of cholera. In the case of a woman who died in the sixth month of pregnancy we found the liquor amnii to be in the usual abundance, but cases we believe, are related where it has been all absorbed.

Purpura.—As a symptom, purpura may be found accompanying various general and local disorders, such as fevers and the exanthemata, as well as diseases of the liver, spleen, &c. In the absence of any such causes, we are obliged to look upon the affection as a specific one, and call it purpura. However, this is not common, but occasionally occurs, and sometimes constitutes a disorder which is fatal in a few days. As during life the whole body is covered with spots, and hæmorrhage occurs from every opening on the surface, so after death all the tissues of the body show effusions of blood, which is found to have burst forth from all parts of them. The serous and mucous surfaces are found covered with extravasated blood, also the bladder and kidneys, and in most cases also the brain; it may escape into the eye, causing blindness; the exudation of blood into the brain is often the immediate cause of death by interfering with important parts of the organ; in other cases death is caused by exudation of blood into the lungs, or simply by exhaustion; in some cases the spleen is found inordinately large, as well as its corpuscles.

Scurvy.—In this disorder the body is blotched with marks of hæmorrhage at different depths and of different ages, the superficial and recent patches being purple, while the older and deeper show the usual green and brown changes. You will commonly find hard brawny tracts generally in the limbs, especially about the knee. A section of these shows them to be due to effused blood, more or less altered; this blood may be in the muscles and intermuscular tissue, and rarely under the periosteum, sometimes in all these parts together. The joints may contain blood; the knee-joint may be distended with it; inflammation of the joint may have arisen as a consequence. Hæmorrhages, similar to those found in purpura, affect the great viscera and alimentary canal, whose interior may be blotched with ecchymoses, some of which may be found eroded. The heart is soft and pale, showing fatty degeneration within in many cases, and there may be embolic infarctions in the lungs or spleen; further, the lungs may show pneumonia; and the spleen is large and soft; the blood has a watery appearance.

Anæmia.—We occasionally meet with cases of fatal anæmia where no disease is found in the body; patients, after being in an almost bloodless condition for some months, die, and all the organs are found pale, and some in a commencing state of fatty degeneration; there is also generally some exudation of serum into the serous cavities, and œdema of lungs and other parts. We have now seen several of these cases; the blood resembled pink water, and formed no coagula in the vessels or heart. The latter organ exhibits in a marked degree that form of fatty degeneration where the internal surface, especially the left ventricle, presents the peculiar mottling from change in the muscular fibre.

Diabetes.—In diabetes the body is wasted, but hitherto no local change in explanation of the cause of the malady has been discovered. The cause of death is generally found in the lungs. The brain has a healthy appearance. Dr Dickinson has described minute degeneration following the course of the small vessels, and revealed by extravasations of blood, and some disintegration of the tissues, with the presence of granular corpuscles; all these he found in the medulla oblongata, cerebellum, pons, or more widely distributed. We have searched several cases, without discovering more degeneration than belongs to wasting diseases. Since the discovery of artificial diabetes by wounding the floor of the fourth ventricle this part has been especially examined, but there are not more than three or four instances of diabetes from obvious disease there; and in these one may say the state observed was rather saccharine urine than true diabetes, conditions sufficiently distinct. The liver, too, whose sugar-forming function (directly or indirectly) has drawn to it so much of the attention of physiologists, generally looks quite healthy; we have generally found it small, sometimes very small, certainly not looking as if it had been overworked; in section quite healthy, but devoid of fat. Diabetes, indeed, has never in our experience coexisted with severe disease of the liver. On the other hand, the kidneys are large, indicative of the increased work they have had to perform, the pair often weighing sixteen ounces to eighteen ounces; we have found the epithelium sometimes loaded with fat—good examples of simple fatty kidney, without any Bright's disease or other morbid condition.

The alimentary canal is generally healthy; the intestines usually filled with hard scybala; we have once, however, seen diabetes fatal by acute ileitis.

The lungs are nearly always diseased; sometimes death has taken place rapidly by pneumonia, which in one of our cases ended in gangrene; but generally the affection is an ordinary phthisis, of the pneumonic or tuberculo-pneumonic kind, vomicæ being present in the upper parts, and caseous pneumonia and tubercles in the lower parts. We have also seen death occur suddenly, without discoverable organic disease.

Syphilis.—We need not speak of primary syphilis, deaths from which are now very rare, but will consider the later affection of the great viscera, which indicates the general character and distribution of the disease, while it produces a cachexia that may develop a lardaceous state of the organs, and so be fatal by anæmia and albuminuria, or fastens on some vital organ, and by interfering with its structure destroys life. We have already, in connection with the separate viscera, spoken of changes and deposits which may be attributed to syphilis, and we will now class them together as a whole. It is only of late years that its effects on the internal organs have been known; even at the present time it would save many valuable lives if syphilitic affections of the viscera were more readily and generally recognised.

If one looks over a series of cases of syphilitic change, and compares these with any other forms of change, one finds that the syphilitic cases have characters by which they are practically easily distinguished. These characters we will attempt to state. 1. Generally a small part of the organ is attacked, and the remainder is left quite free. The disease is strictly localised in the spot it affects. 2. Its outer part is composed of fibrous tissue, which can be seen to represent the natural fibrous supporting elements of the part in a state of augmentation, while the functional elements of the part have dwindled away. It is a local sclerosis. 3. Its central part shows the now celebrated, caseous or gummatous, faint yellowish matter of more and more elastic consistence, and less and less friability and curdiness; generally rather sharply distinguished from the fibrous outer part, and sometimes softening down or calcifying. 4. There are signs of more acute inflammation in the immediate neighbourhood, showing lymph, &c., or adhesions to the parts around. (3 and 4 may be absent.)

Such patches, sharply contrasting with more healthy tissue immediately about them, and (5) distributed more or less widely in a variety of organs, but especially in the testes and liver, are not a general thing that could be passed over as a common accident. Their characters attract attention. A syphilitic gumma in muscle or brain is so unlike anything else that, if seen for the first time by one who knows the rest of the common run of pathological changes, it demands from him some recognition of its peculiarities. In short, it is not common, but specific in the strict sense of the word. We mean that we are not to suppose that these syphilitic changes are of a kind that are otherwise recognised as non-syphilitic, or in any sense of a "common" kind. It is not so. They are quite peculiar, and strike the eye and the mind as peculiar. Allow that they have characters like the verbal stock descriptions of "inflammation generally" (a thing which has no existence); the commonest inflammation has its common peculiarities. These characters of common inflammation are not the characters that we rely on to know them by; the five characters we have above enumerated do not belong to any other change, and amply suffice to distinguish practically the syphilitic from all other changes.

The general result of observations of the structural characters of syphilitic deposit in all parts of the body is the same, and to this effect, that there are few now who suppose that syphilitic formation presents any characteristic *elements*, or that the absence of characteristic elements invalidates the evidence of the syphilitic nature of these formations.

It is well to remark that the uncertain nature of the elements might have been due to their never having any characters sufficiently defined to distinguish them from any imperfect or indifferent cellular elements, so that the examinations of a syphilitic tumour might not have ever revealed any elements with *character* about them. Just as formerly tubercle was described as being made up of "ill-defined," or "imperfect," or "degenerate," or, as it is sometimes put, "poor wretched" cells. But, in fact, the difficulty of recognising syphilitic elements arises rather from their variety of character than their want of character.

In truth, we often find syphilitic formation whose elements are in a high state of development. Thus, in a case of syphiloma of the dura mater that occurred the other day, the elementary cells would, we think, have been called "cancer" cells. Some that were pointed to under the microscope showed the possibility of the syphilitic cell reaching a large size, and having a bold nucleus with double contour, and provided with distinct nucleoli; indeed, just such an element as responds to the ordinary futile account of a "cancer cell."

But, on the other hand, syphilitic deposit often does not show these defined and well-characterised cells; it may even offer nothing more than common wavy areolar fibrous tissue, more or less deformed, with some fat-grains embedded, or some decayed nuclei present in it. These differences are partly a matter of stage or *period of growth*. The large and better-formed cells being new and young, the old fibrous and fatty material being at the period of full development, or senescence, or decay. We nowhere else have so good a reason to think the senescence and decay of formed elements may be due to medicines. These varieties from stage give so much diversity to the characters of the matter formed through syphilis, that some of them may easily be misunderstood by observers who know others tolerably well. To the naked eye the formations offer a like variety. Thus, in the liver an isolated softened red patch is the first stage; it is sometimes met with in cases where the next stages identify it. This red patch is, in the second stage, found to have a pale yellowish sub-elastic tough substance forming in its middle, while around this substance, within the red patch, there is recent fibroid formation of the usual greyish cast of colour, the remains of the red soft patch encircling and spreading around these. Then, a third stage, a softening or a calcification of the yellowish centre, proceeds, or else a simple wasting away of it, which latter is more common; so that there usually remains at last a sunken scar in the place of the tissue, which has in this way been changed and removed. Such a series of states from the red patch to the calcified, softened, or merely scar-like relic, gives scope for great differences in the actual appearance of individual instances, and these stages in themselves would make syphilitic formations various enough. But we believe that all the differences in syphilitic deposits are not due to the stage or period of the growth.

In some cases the syphilitic formation wears very much the appearance of actively growing tumour. This is especially the case in the membranes and cortex of the brain. It forms a large and bold mass with rounded outline and fleshy consistence, enclosing in the middle some caseous change, but with a considerable proportion of it vascular and fleshy, so that from the appearance alone it could not be told whether the disease were a sarcous tumour or not; and one has to view the whole case and make a judgment, or at least arrive at a conviction, from the general characters of the other formations in the body, the history, &c. Such a case occurred at Guy's about five weeks ago. The difficulty in post-mortem recognition was at first considerable.

In the opposite extreme, the syphilitic formation appears in the shape of small grains, like tubercles, or as diffused yellowish soft material in the membranes of the brain, accompanied by the signs of inflammation in these. Such a case we published among a series of cases of syphilitic disease of the viscera in the 'Guy's Hospital Reports' for 1867.

The difference between these tubercle-like granules and the tumour-like masses is more than one of stage. To realise the relation of syphilis to the former, we may best suppose that a meningitis is qualified by the syphilitic poison, so that characters of diffuse inflammation and of syphilis are combined, as we often find tubercle combined with the products of acute inflammation. In the other case, it is perhaps true that the disposition to form tumour in the part is similarly qualified by the syphilis, so that the product is syphilitic tumour. We do not intend these suppositions to be explanatory, but only to assert a belief that, whatever the causes of inflammation or of tumour may be, they can be determined into syphilitic character by the presence of syphilis in the system.

We need not again detail to you the special appearances of syphilitic disease of the several organs and systems, as we have already described them when speaking of the organs themselves. When syphilitic formations are present you will often find lardaceous disease of the abdominal viscera.

Lardaceous Disease.—We have described the lardaceous condition of the several organs especially liable to it; in all of them the change is essentially the same. It first affects the minute vessels, especially the arteries, and extends to the capillaries, afterwards invading the proper elements of the texture implicated. Its favorite seats are the kidney and spleen, which are about equally often lardaceous; the liver, which stands near them in liability; the lymphatic glands, which are frequently involved; the alimentary canal, which is not so generally obnoxious, yet often gives evidence of lardaceous disease, and is prominent on account of the obstinate symptoms which its disorder induces. After these come parts which have certainly much less attraction for the amyloid matter, yet when it reaches them, obtain a very decided charging with it. Such are the supra-renal bodies, the thyroid gland, and the tonsils. Following these, very far from them, are a number of other parts, such as the muscles, the membranes of the brain, the bladder, serous membranes, &c., even to the nerves of the heart and of the uterus. Lardaceous change is found in many books in these situations, but we can assure you it must be very rarely indeed discoverable in real muscles, membranes of brain, &c. We have carefully searched in cases where the disease was excessive in its proper haunts, yet have not been able to detect it elsewhere; once we found a low degree of it in the small arteries of the diaphragm, and we have seen it in epithelium from the renal pelvis and bladder discharged during life, also in the deep and new-formed epithelium on thick-skinned feet with elephantiasis or lupus. You will find a little iodine added to the glycerine or syrup you use in microscopic examinations of fresh specimens very useful in detecting such changes, as well as in defining the outlines of texture elements. To avoid errors you must make yourselves familiar with the port-wine-like red tint of the iodized lardaceous matter as seen by transmitted light. This has evidently been often the subject of mistake.

As to the nature of the change, the peculiar reaction with iodine still remains almost the only distinctive or well-ascertained feature of the morbid process. In the 'Report of the Path. Soc. Committee,' 1872, Dr Marcet says that iodine has the power of precipitating an aqueous infusion of a lardaceous liver, throwing down a dark matter, to which matter he ascribes the deep colour produced on lardaceous organs by the action of iodine. He found that ultimate analysis of lardaceous parts showed less of nitrogen than is present in albumen, a fact which would rather argue that the amyloid substance is, after all, of the nature of a ligneous or starchy matter; you must remember that any lardaceous tissue used for such analysis contains only a small relative part of the amyloid substance, inseparably engaged in the nitrogenous proper texture of the tissue. We have already alluded to Dr Dickinson's hypothesis as to the nature of the amyloid material (page 435).

We have never seen any facts to show that lardaceous change leads to a fibrous scar-like withering, as some have described; and we believe the scar-like patches in question to have been due to syphilis, a frequent concomitant and cause of lardaceous action.

Amongst the causes of lardaceous disease syphilitic and scrofulous disorders of the bones are most prominent. Chronic suppurative bone-disease is so frequently the cause that one naturally examines the bones to ascertain if the matter has arisen there, but the bones do not then yield the amyloid reaction. It has been said, however, that the lymphatic glands corresponding to the diseased bone are first implicated; this we are not able to confirm; we have several times found the corresponding glands free when the viscera were much involved in the disease. Late syphilis without any suppuration will certainly produce profound amyloid change. So, it is said, will malarious fever, but there is, we think, suspicion that syphilis complicated the cases published by Frerichs in proof of this; the description of the liver strongly infers syphilitic disease in those cases. Otherwise the amyloid affection has almost always attended upon prolonged suppuration, such as that in phthisis, in scrofulous disease of the kidneys, in old dysenteric ulceration, in old obstinate carbuncle, leprosy, &c.

The amyloid reaction with iodine is not restricted to the kind of disease we have just been considering, but it is present in certain corpuscles found in the neuroglia of wasted brain, &c. These corpuscles are further interesting in this relation because they very closely resemble starch-corpuscles in their rounded figure, and even in possessing a hilus, in some instances with concentric markings. We have already alluded to the distribution of these corpuscles in softening or sclerosis of the nervous centres. All of the bodies in question are not tinted by iodine; you often find them uninfluenced by it, and these corpuscles indifferent to iodine come near towards the likeness of the rounded colloid corpuscles in the prostate, also towards others less closely resembling them in blood-clot, and towards the amyloid matters of diseased growths, &c. The discovery by Schmidt of cellulose in the tunics of certain ascidian molluscs furnishes a physiological type for these pathological changes. The idea suggested by Meckel, who supposed that amyloid reaction is due to cholesterine, is not admitted, because the solubilities and other chemical attributes of amyloid matter and of cholesterine differ essentially from each other.

Burns.—Persons not destroyed by the heat and suffocation, often, if the burn is severe, die in the course of twenty-four to forty-eight hours. The destruction of a considerable part of the skin will then be seen, but usually the viscera are found to all appearance healthy; or there may be in rarer cases evidence of bronchitis. In cases that survive several days you may find broncho-pneumonia, or catarrhal nephritis, or deep ulceration of the duodenum, perhaps opening large arteries or perforating the peritoneum; more rarely some other part of the alimentary canal may be ulcerated. Yet later the usual morbid anatomy of pyæmia may be found.

Suffocation, Strangulation, Drowning.—In death from any of these causes the surface of the body shows marked lividity, the great viscera are found loaded with dark blood, and there may be some effusion of blood on the brain, or in the air-passages and lungs, or under the pleura. The blood is everywhere liquid, as usual in sudden death. The heart is generally contracted firmly, so that its left ventricle is empty, while the right cavities, which close less completely and are thinner, contain a larger quantity of fluid blood; there is much of this fluid blood in the large systemic veins. You should make this observation before removing the lungs from the body, but not before you have become familiar with the amount usually present in ordinary cases. Froth may be present in the trachea in death from any of these causes; this froth may be tinged with blood. Thus, the results of internal examination in such cases, which are often separately enumerated as though distinctive, will, so far, not enable you to distinguish between these forms of death, nor, indeed, to distinguish between them and death from epilepsy, &c. Whence it is the more necessary that you should watch keenly those incidental conditions of and about the body which may enable you to come to a conclusion as to the cause of death.

Death by suffocation.—The suspicion of death by suffocation will induce you to thoroughly examine the whole tract of the breathing passages; the nasal cavities being carefully inspected for foreign bodies in some form of dust, which may have been drawn in, and, if found, may indicate the circumstances of the accident. The tongue, too, which often protrudes during suffocation, should be looked to; its tip may be found covered with dust, &c., although now it may be withdrawn into the mouth. The fauces should, of course, be carefully examined. We have met with a case where a man was brought in dead from a public-house, and there was no other disease than a monstrous lump of half-chewed steak, quite filling the upper part of the pharynx. In cases of cerebral disease, especially in softening of the pons, such choking may be found. In suffocation by poisonous sewer-gases the pupils have been found dilated. When the bodies are found some hours after death, and have not received the customary nursing attentions, the eyes are, of course, usually found open.

Strangulation.—In death by strangulation the mark of the cord or other means of compression forms the only ground of diagnosis afforded by the body. The marks correspond pretty closely with the points of rough contact of the fatal means. In hanging they are found chiefly behind the ear, about the occiput; in strangling with cords they encircle the neck; pressure with the fingers leaves the imprint of these. Such marks are not absolutely always, but are pretty generally present. The marks themselves are seen in a dirty yellowish dryness of the spot, which is hardish, and has partly lost its epidermis. This is the case if the body has lain many hours, but at an earlier period, or in slighter degrees of injury, the mark may be livid, with folds, creases, &c., printed by the ligature; blood may possibly be effused beneath it. Experiment has shown that all these effects may be produced by suspension or ligature, on bodies recently dead. In some cases of *hanging*, great damage is done to the deep textures, the neck being dislocated or broken and the soft parts torn; but death more frequently, in suicidal cases, results from asphyxia.

Drowning.—In the examination of drowned persons it is very important to note minutely the state of the surface of the body, observing any scraps of weeds, mud, &c., that may be found under the nails or clinging to the surface; also to inspect very carefully any apparent bruises or other injuries. All this, with the state of the surface of the body, showing the effects of continuous soaking in water, and the laws that determine decomposition, according to the period of the year, and the running or stagnant condition of the water, will belong more naturally to the compass of works on jurisprudence, to which we must refer you.

In the internal examination you look for the presence of water in the air-tubes. It has been disputed whether the water in which the person is drowned finds entry, but this undoubtedly occurs. Also in the stomach and in the œsophagus, such water, having in it, perhaps, weeds, &c., may be found. In shifting and carrying the body the contents of the stomach may find their way into the windpipe; this can generally be known by their strongly acid reaction. It appears from some carefully recorded cases that death may take place through shock, when persons are thrown or fall into the water, so that the characters of asphyxia are not produced. The same remark applies to deaths by suffocation and strangulation, for in these you do not always meet with the lividity and general congestion which you would anticipate. Persons saved from drowning after falls in the water often die in a few days from acute pneumonia, as you might expect from the congestion of the lung, often so great that hæmoptysis follows, especially remembering the water breathed into it and the chill and the shock which the person has undergone.

Death from Lightning.—Bodies of persons struck by lightning generally show marks of more or less severe injury in the form of contusions, ecchymoses, lacerations, burns, red streaks, &c. The bones may be broken or the viscera lacerated in some cases.

Sunstroke.—We have examined but few cases of sunstroke, and the result was as unsatisfactory as that generally arrived at, namely, nothing whatever distinctive of the condition was found. There was a general state of congestion that is usual in death from coma.

Gout.—The morbid anatomy of gout will be found described under Diseases of the Joints (page 79), Heart (page 132), and Kidneys (page 509).

Leukæmia is described under Spleen (page 476).

Poisons.—The effects of poisons, so far as they are anatomically evident, we have described under the several viscera affected by the poison. The consideration of such disorders in their character and grouping as effects of special poisons we think belongs rather to medical jurisprudence.

TUMOURS

The word tumour signifies simply a swelling; and there, perhaps, was a time when that was all that was meant by it, but every swelling is not now called a tumour. Thus, the common unsightly swelling of the face, from carious tooth and abscess, is never called a tumour. Yet the exactly similar appearance caused by growth from the gums or facial bones is a tumour. Again, ascites is never known as tumour, and that, not because it is better known as dropsy, for the limited dropsy of hydrocele is called a tumour. So that swelling is not what is meant by tumour, indeed some of the most dreaded tumours often shrink the part they attack; as, for instance, scirrhus of the breast and certain puckering cancers of the peritoneum.

A strict definition of the word tumour is, perhaps, impossible. In fact, a pathologist trying to define a tumour is like a botanist trying to define a shrub; for just as the word shrub was never meant to be botanical or scientific, being simply "what would grow in a shrubbery," so "a tumour" was never meant to be a pathological or scientific term; it means an obstinate, persistent, unmanageable swelling, that wants removing if possible.

The kinds of swelling which it is agreed to call tumours have the following set of characters:

1. They arise by a new centre of growth.
2. They organize in solid continuity as they grow.
3. They persist.
4. They form circumscribed, or, at least, easily determined swellings.
5. They are abnormal (or the above characters would include the fœtus, if not the several organs of the body).

Now, this is, we think, the best way of solving the question, "What is a tumour?" The *beau idéal* tumour would include all these characters. Such a tumour as an adenoma of the breast, or sarcoma of bone. No one would hesitate to give the full force of the word tumour to such a growth as possessed all these characters.

But many things that lack one or more of these characters are still called tumours. Thus—

1. Some tumours have no new centres of growth, but are enlargements of existing parts, such as splenic and glandular tumours.
2. Some tumours do not undergo organization, such as ranula, wens hydrocele.

3. Some shrink the part they grow in, such as scirrhus and peritoneal cancer.

So that many of the things called tumours correspond but partially to the standard of the most perfect tumours, which develop from centres, and organize themselves like new organs in the frame; and thus they lose many of the characters of those perfect tumours, until you reach such structures as syphilitic gummata and large tubercles, which you would scarcely call tumours, and yet hesitate to deny the name.

As to the cause of the origin of tumours, you may expect us to say something, but this question rather belongs to theoretical pathology than to morbid anatomy. We will only enumerate to you four views which have in a more or less pure and clearly conceived form prevailed at different times or with different authorities. These views are as follows:

1. There has been thought to be a state of mixture of the blood, such that the substance of the tumour comes out as a *residue*, when the other organs have taken their respective shares of nourishment.

2. An organizing power in the *blood*, so that it actually pours out material that forms a tumour.

3. A growing power in the solid elements of *texture*, which creates the tumour by augmenting such elements.

4. Lastly, there has been recognised a kind of *parasitic* nature in the life of the new growth.

We do not mean that these views came in such an order of succession in time. For we believe that in one form or another they are always to be traced coexisting, and that even at this day we must allow these four kinds of view to be no more than so many just and necessary acts of recognition of certain phases of tumour formation, and that it is still a question what their relative importance may be.

Indeed, each of these views has its separate history, though the histories overlie and are intermixed with each other, for, unfortunately, although they really are quite distinct, yet their distinctness has not been borne in mind during the long-continued discussion on the general subject of tumours. However, it is not our intention to enter into this discussion. If you feel disposed to go further into the question we must refer you to a paper in 'Guy's Hospital Reports' for 1872.

As the different kinds of things called tumours have so little in common, general remarks upon them are not of much value. So we will proceed to describe to you the leading structural features of the various special kinds of tumour as they are now generally recognised.

Some authors attempt to gather these kinds of tumours into larger groups, but with little success. There is, however, a very general recognition of some such classification as this:

1. Cysts.
2. Tumours with the structure of a simple tissue, or *histioid* tumours, as they are called.
3. Tumours of the structure of a compound tissue, or *organoid*, as they are called.

But although this division appears clear, yet the application of it is subject to much serious discrepancy, so that it is practically worthless; thus, among cysts, those known as dermoid cysts are, perhaps, the most organoid of all tumours. Again, osteoma, or bony tumour, which is classed as histioid, that is, composed of a single tissue, really contains, as you know, fibres, blood-vessels, and generally cartilage, besides its proper *tela ossea;* while, on the other hand, angeioma, or nævus, which is called organoid, only contains some areolar tissue along with its proper dilated vessels, so it is really no more organoid than bone. Yet again, some authors regard carcinoma as a characteristic organoid tumour, while others deny it that name, and include it with sarcoma under a fourth kind, which they call *cellular*. Hence, as the classification is involved, and not worth discussing, we do not adopt any such division of tumours into greater groups; but will only distinguish them into their leading kinds. For convenience of description, however, we will group the several kinds of tumours as follows. The reasons for such grouping will sufficiently appear in the course of what we have to say upon them.

1. Cysts.
2. Osteoma; osteoid chondroma; enchondroma.
3. Fibroma, sarcoma, glioma, myoma, psammoma.
4. Myxoma, lipoma, myxochondroma.
5. Adenoma.
6. Carcinoma.
7. Lymphoma.

Cysts.—Some cysts own a very simple origin; they are mere enlargements of natural cavities or channels. Thus, natural cavities may be distended with fluid; for instance, that of the tunica vaginalis, which then forms the cyst called hydrocele. Such cysts are distinguished by Virchow as *exudation cysts*.

The channels of ducts becoming obstructed, a dilatation of the duct or of the follicles of the gland behind the impediment occurs, and then are formed *retention cysts*. Numerous instances of these, such as wens, ranula, &c., will rise to your minds.

Hæmorrhage also gives rise to cysts, as in the external ear in the so-called hæmatoma auris (see Ear), in the brain, in the substance of tumours, &c. These have been called *extravasation cysts*.

Other cysts arise in tumours by the softening down of the texture; these are not unfrequent in enchondroma, myxoma, fibroma, sarcoma, adenoma, and carcinoma. The resulting cavity contains at first a turbid, often mucoid fluid, but this becomes clearer afterwards and the walls of the cavity may become quite smooth—*degeneration cysts.*

Besides such exudation, retention, extravasation, and degeneration cysts, whose origin is capable of being demonstrated, there are other cysts which appear to arise spontaneously as new growths.

Some others appear as enlargements of the remains of fœtal organs, such, for instance, as encysted hydrocele of the testis, cysts of the broad ligament, &c., and dermoid cysts.

Others are overgrowths of the natural elements in a tissue, such as the Graafian follicles, and, according to some views, a variety of renal cysts.

Others yet appear due to distension of the areolar tissue spaces, as encysted hydrocele of the cord, cystic myoma of the uterus.

Again, dilatations of the lymphatic channels form tortuous spaces in the hypertrophied subcutaneous tissue, as of the thigh occasionally.

Osteoma, Osteoid chondroma, Enchondroma.—These compose a sufficiently natural group of kinds; indeed, it is often practically difficult to refer a tumour to one of these kinds, they are so apt to be mixed together in the same tumour. However, when bone forms a large part or apparently the whole of a tumour, the tumour is called an *osteoma*, but no tumour is ever really formed altogether of bone; there is always present an ossifying matrix, by the ossification of which the bony part of the growth enlarges. So that the growth is not of bone, but of the soft part which precedes it, and which *changes to bone when it has already grown*, and only so far as it is not any longer growing. The kind of matrix varies much, and the structure of the whole growth takes its vital properties from the matrix. Thus, sarcomata or even carcinomata may directly ossify, and so we may get *osteo-sarcoma* and *osteo-carcinoma*. But when this is so the bone is in relatively small amount, and of still less comparative importance; the tumours are practically sarcoma and carcinoma, and the kind of interest that attaches to the bone in them lies rather in this, that such tumours arise almost always from bone, and we see in the bony development an evidence of the continuance of the nature of the mother tissue into the nature of the offspringing tumour.

The forms of matrix which produce growths of practically a bony nature are generally two, viz. fibre or periosteum, and cartilage. Periosteum—or, to speak more exactly, a tissue resembling closely the deeper layer of the periosteum—forms large tumours, whose transformation into bone takes place in the manner shown in the left side of Pl. I, at *a*, *b* and *c*; the cells take the shape of bone-cells and the matrix calcifies; these tumours are called *osteo-fibroma*, *osteoid chondroma*, or *periosteoma*.

Cartilage forms large tumours, generally arising from bones, especially those of the extremities, and more especially of the distal parts of the extremities; these grow generally within the bone, more rarely under the periosteum. To the unaided senses these tumours offer plainly the well-known characters of cartilage, except that they are nearly always divided by septa into lobules; the consistence varies, and when large they may be in parts very soft and sticky or mucous or softened into cysts; when this is so their structure varies towards that of mucous tissue, whence they are called *myxo-chondroma* (Pl. III, *b*), but instead of softening, a large enchondroma often hardens, and appears to be ossified, though it is only petrified by deposit of calcareous salts in its matrix (see Pl. I, *e*); this change is, as is well known, the first step in ossification of cartilage. In many cartilage-tumours the process goes no further, or it may proceed through the several stages shown in the right side of Pl. I, viz. vacuolations (*f*), formation of medulla-cells in the vacuoles (*g*), and direct transformation of these to bone-cells (*h*), as seen in the lower and right part of the drawing. More rarely the cartilage-cells, without calcifying, proliferate and change directly into bone, as seen in the middle of the figure (*i*).

The amount of cartilage, periosteum, or bone present varies indefinitely in different cases. When cartilage preponderates, the tumour is called an *enchondroma* (Pl. I, *d*; Pl. III, *a*); when bone preponderates, an *exostosis*, *osteoma*, &c., according to its shape and connections (page 51); when periosteum preponderates, an *osteoid chondroma* (Pl. I, *b*), as before said; if the ossifying tissue is simply fibrous, it is called *osteo-fibroma*.

Occasionally in tumours that project from the surface of the bone, as of the femur, &c., the amount of bone and cartilage is so equal that it is a matter of difficulty to decide which name shall be used, and then the terms *cartilaginous exostosis*, or *ossifying enchondroma*, are employed. *Osteoid chondroma* is to be suspected of malignancy; it makes a part of what were called *osteoid cancers*, being liable to set up similar growths in remote parts, as the lungs, &c.

Fibroma, Sarcoma, Glioma, Myoma, Psammoma.—These form a natural family in as much as they correspond in structure to the fibrous and connective tissues, including those forms of connective which enter into the composition of the organs, and particularly the neuroglia or connective substance which unites the proper nervous elements in the brain.

The distinctive character of all these kinds of tumour is the possession of a structure microscopically homogeneous, and consisting of cells with an intercellular matter surrounding them, so as to be between them all. The qualities of this intermediate tissue between the cells determines the kind of tumour. Thus, in *fibroma* it is fibrous (Pl. I, *a*), in *sarcoma* it is soft and slightly fibrillated (Pl. II, *h*), in *glioma* it is yet softer and more delicate (Pl. II, *a*), being sometimes granular and almost fluid, at other times showing a felt-work of extremely slender long fibres. The proportion of cells varies extremely; sometimes there are few, as in some fibromata and gliomata, which are almost wholly fibrous.

The larger the cells and the greater the proportion of the whole tissue they compose, the more is the sarcomatous character developed. To understand the relation of sarcoma to the other kinds, we must remember that in all the connective class of tissues the character of the tissue depends on the intercellular matter. This is strongly fibrous in tendons, more delicately fibrous in areolar tissue, imperfectly fibrillated or quite homogeneous in the papillary layer of the corium, extremely soft in the brain, &c. The cells within it in these normal tissues, are so few as commonly to escape notice, but in the formation of tumours corresponding to either of these kinds the cellular elements increase disproportionately, while the intercellular substance is not only thus relatively less in amount, but also is deficient in character.

Now, when the increase of cells and alteration of the intercellular substance are not so great but they still leave the main characters of the tissue recognisable, the tumour is called after the tissue it thus resembles, a fibroma, or glioma, &c. But when one of these tissues is produced very rapidly it has no time for its intercellular matter to acquire the proper characters of its normal structure, and hence it remains intermediate, while it also is small in quantity, the cells greatly preponderating. This constitutes sarcoma; indeed, you will find that *any of the previous group of tumours (osteoma, &c.) will likewise become sarcoma by rapid growth of the cellular elements* and deficient development of the intercellular matters, and thus osteoma becomes osteo-sarcoma, &c.

In this way any of the normal connective tissues may produce by rapid development a tumour of sarcous tissue or sarcoma (the name is well chosen, σαρξ, which equals *caro*, or our word *flesh*, means commonly any soft animal substance, not blood nor bone). It follows that while yet the character of the original tissue is recognisable in the tumour this will qualify it, so that there will be several kinds of tumour approaching sarcoma according to the tissue of which each is a development, while the condition of pure sarcoma is a culmination of the rapidity of development, such that all character of the intercellular substance is lost.

These tumours, as we have said, are named according to the tissue they are derived from or resemble. Thus, those from lymph-gland are called lympho-sarcoma, those from the neuroglia glio-sarcoma, those from the fibrous and areolar tissue fibro-sarcoma, and those from bone osteo-sarcoma. In either case, when the character of the original matrix is lost, they are merely called sarcoma.

The principal of these kinds are seen in the schematic figure, Pl. II. The round-celled kinds generally arise from lymph-gland, or neuroglia, or mucous tissue; hence they are common in glio-sarcoma (Pl. III, *a*), or lympho-sarcoma (Pl. II, *b*; VII, *g*), or myxo-sarcoma (Pl. II, *c*, *d*), though less constant in the last. The spindle-celled kinds arise from connective, fibrous, or bony tissue, and hence they are common in fibro-sarcoma (Pl. II, *e*) or osteo-sarcoma.

Some sarcomatous tumours contain very large polynucleated cells (Pl. II, *f*), more or less rounded in outline, embedded among spindle-cells of usual character. These cells are known as giant-cells, they are most characteristically developed in some examples of epulis and tumours of medulla of bone, to which we refer you (page 362; see also Myeloid).

You should observe, in microscopic observations of various sarcomatous tumours, that cross-cut bundles of spindle-cells appear as groups of round cells, so that it requires careful focussing to distinguish one from the other (Pl. II, *g*, *é*).

The figure (Pl. II) is composed of accurate drawings of portions of the several kinds of sarcoma named, but they are gathered together in a diagrammatic way, the forms being graduated into each other as they appear when found side by side in the same tumour. You will soon notice, in examining specimens of such tumours, that several of these varieties of structure are apt to be found in different parts of the same tumour.

Tumours of this group appear to the unaided senses either as evidently resembling the tissue from which they are named, or more or less soft and fleshy in proportion as they approach the condition of pure sarcoma. As they deviate from their normal type they tend to be subpellucid and elastic. They yield no milky juice when the section is scraped, as you will find that carcinomata do.

Glioma (Pl. II, *a*), or tumour of the neuroglia, is almost peculiar to the brain and nerves, in which association we have already described it.

Psammoma, or sandy tumour, is a name given to certain rare growths, in which calcareous salts are found in the fibrous basis of the tumour, making it gritty or even sandy in section. They are almost limited to the membranes of the brain and spinal cord.

Myoma resembles sarcoma, but the cells develop into non-striated muscular fibre-cells. These tumours are almost strictly limited to organs of which non-striated muscular fibre forms a considerable part; they are especially frequent in the uterus and prostate, but may be found throughout the alimentary canal occasionally.

Melano-sarcoma is a name applied to sarcomatous tumours which are coloured brown or blackened by a deposit of pigment in their cells. Such black tumours generally arise in parts where pigment is naturally present, as in the eye, the pia mater, and the skin. Except for the blackness, the tumours have the ordinary characters and tendencies of sarcoma.

Myxoma.—The name myxoma is given to all tumours of connective-tissue type which contain mucus or mucin in their intercellular matter. It corresponds nearly to gelatinous sarcoma, collonema, and fibro-cellular and some colloid tumours of older authors. The forms of the cells are very variable, but in the most typical examples of such tumours, and especially in their older and fully developed parts, the cells are large and usually multipolar or "stellate," with a distinct nucleus and nucleolus (Pl. III, *c*); the stellate branching rays of the cells are mutually connected, so as to form a more or less open network, in the interstices of which the mucous semifluid lodges. Beams and bands, which generally have a stiff rigid appearance and an angular rather than a wavy disposition, pass about, dividing up the substance of the tumour into very imperfectly defined sections, more or less visible to the naked eye; from these beams arise fine fibrils continuous with the cellulo-fibrillar network. Cells can be seen embedded in the beams themselves, which may preponderate so as to give a fibrous character to the whole, acquiring the name *myxo-fibroma* (Pl. III, *f*). In any myxoma much of the tumour, and especially the younger part, may be found formed of spindle-cells (Pl. III, *d*). These are really connected, by means of threads from their sides, the threads joining together, and thus all that is required in order to produce the stellar figures of the typical and complete myxoma cells is the drawing out of these threads to greater lengths, through the separation of the texture-elements by an increasing quantity of mucus infiltrated into the tissue, and thus the compact spindle-cell parts are young, and the stellate parts middle aged. In yet other parts the prevailing form of the cells is round, or with one pole (Pl. III, *e*); and these round cells resemble ordinary mucous corpuscles, like these being scattered in the mucoid matter, while the stronger fibrils still remain; such cells often contain many fat-grains, and are found in the oldest parts of the tumour, representing the senescence of the growth by fatty decay of its cells.

Thus, there are different degrees of softness in the several portions of a large myxoma, according to the various ages of its parts; the younger portions being more compact, and the older showing a greater quantity of mucus, until some parts may appear clear and pellucid, when the term *hyaline myxoma* (Pl. III, *g*) is applied; or such soft parts may be turbid through the fatty cells present, while they are still gelatinous and tremulous in consistence. The softness of the fibres and cells may go on until a cyst containing mucoid fluid is formed; such a tumour would be called *cystic myxoma*. Although we so far describe the variations of consistence as developmental, and representing stages in the life of the tumour, some growths, undoubtedly, show a greater tendency to the breaking down of the solid web of cells and fibres, while others have naturally a much greater proportion of solid part in the form of strong bundles of fibres, which may make a close tough tumour, only known to be myxomatous by its stickiness; this would be called myxo-fibroma, as is said before.

Other variations in the nature of the tumour may take it towards the characters of cartilage, thus producing *myxo-chondroma* (Pl. III, *b*), the cells becoming encapsulated and the interstitial substance chondrinous; or the cells may enlarge and fill with fat until the appearance approaches, or is identical with, that of adipose tissue, so forming *myxo-lipoma* (Pl. III, *h*). In other examples you may meet with large polynucleated cells, like those called giant-cells (Pl. III, *k*), which more specially characterise the kind of sarcoma once called myeloid; these are, however, rare.

Myxoma generally forms large soft tumours of elastic consistence; they are more gelatinous and tremulous in their consistence than sarcoma; the section is sticky, and its appearance generally more pellucid than that of sarcoma.

In the theory which supposes each tumour to have a type in some normal structure, myxoma is affiliated to certain natural tissues, in particular the jelly of the umbilical cord, the vitreous of the eye at a stage of its development, and the early stages of adipose tissue, as well as a stage of bone formation out of cartilage. It will be seen that these typical tissues are only transitory in their nature, as compared with such stable tissues as bone, cartilage, tendon. It is in accordance with this instability of their type that myxomas themselves should show transitions to permanent kinds of connective tissue, such as fibrous tissue, cartilage, or fat, as we have already mentioned. Tumours are not unfrequent, especially in the parotid region, which are thus intermediate between cartilage and mucous tissue (Pl. III, *a*, *b*), so that one cannot say to which they most properly belong; also many fatty tumours show clear gelatinous patches of mucous tissue in all transitions to fat, while some myxomata show opaque spots composed of true adipose tissue (Pl. III, *i*).

Adenoma.—The essential character of adenoma lies in the possession of a glandular structure (Pl. IV, *e*); but the comparative amount of the glandular element varies much. There is also variety in the kind of tissue which is found between the gland-follicles (see Breast). Some tumours show structure identical with that of compound racemose glands, more commonly the follicles or ducts are dilated more or less, so as to form cysts (Pl. IV, *d*); one or more of these may prevail, so as to give a cystic character to the whole (*cystic adenoma*). Besides the cysts arising in this way, others may be formed by a breaking down of the intermediate tissue, especially if it happen to be mucous tissue. But such pure adenomata are comparatively rare; it is far more usual to find the glandular elements surrounded and separated by a new formation, which may be so much developed as to more or less entirely take away the glandular character of the growth; this interstitial tissue may either be fibrous, sarcous, or mucous, constituting *adeno-fibroma* (Pl. IV, *a*), *adeno-sarcoma* (Pl. IV, *b*), or *adeno-myxoma* (Pl. IV, *c*), or more rarely it is cartilaginous or areolar; or it may present characters combining these or mediate between them. When the proportion of gland is small, there is doubt whether it is not part of the original gland-tissue persisting in the new substance. Thus, you will see that in an adenoma, &c., the relative augmentation of the cavities of ducts or follicles may make the tumour take the character of cyst, or the relative augmentation of the intermediate tissue may make it take the character of sarcoma, myxoma, or fibroma, so that it may become doubtful to which class you should refer the tumour. But a much more important ambiguity has been introduced because of the likeness of the microscopic structure of adenoma to that of carcinoma; this is so close that some observers class them together. We have, in speaking of the tumours of the breast, discussed this question, and concluded that the difficulty can only arise by too exclusively regarding the microscopic structure of the growths. Nay, that their likeness is rather in verbal description than in reality; for in adenoma the substance of the tissue is just that of gland-follicles, with regular epithelial lining and delicate fibrillar stroma between, while in carcinoma the epithelioid cells are not like gland-cells, but are of large size and various shapes, and have great nuclei with many nucleoli; the stroma, too, is generally different from that of adenoma, being composed of great spindle-cells, or more frequently of an areolar tissue highly charged with small roundish cells; but the chief difference is this, that the unaided senses can already detect the extension of the carcinoma to the surrounding tissue, and the invasion of the lymphatic glands, &c., which constitute its malignancy.

Carcinoma.—This name is now restricted to tumours whose histological structure is composed of an alveolar meshwork of fibroid tissue, the openings of which contain collections of epithelioid cells, filling up all the openings of the meshwork, and so composing a complementary epithelioid meshwork. Thin sections of such a structure show areolæ bounded by a fibroid substance, the epithelioid cells appearing within the areolæ lying close together without any intercellular substance. But a section of any secreting gland, such as the kidney, would show you in like manner a fibroid stroma forming areolæ, in which epithelioid cells are lodged. These characters then are, so far, common to the structure of gland and carcinoma; we have already said that adenoma is the name given to a tumour which has the structure of gland, and hence adenoma and carcinoma are, so far, alike.

But the difference between them is in the comparative perfection of pattern in the adenoma as contrasted with carcinoma. The structure of gland shows regular alveoli composed of a fibroid structure of areolar tissue, and containing epithelioid elements which are of uniform shape, and have small nuclei (Pl. IV). But in carcinoma the alveoli are very irregular in form, and the substance composing their walls contains many spindle-shaped (Pl. V, *d*) or rounded (Pl. V, *e*) nucleated cells. Also the epithelioid contents show cells of very various sizes and shapes, with large nuclei (Pls. V, VI) and bright nucleoli, signs of active multiplying power.

These differences between adenoma and carcinoma are very important, because they correspond to a great difference in their history, carcinoma being malignant, so that it tends to spread in the surrounding tissues and to invade the glands and infect the great viscera remotely, while adenoma is a non-infectious growth.

Five leading types of carcinoma may at present be conveniently distinguished:

1st. Those in which the fibrous meshwork preponderates and the epithelioid contents are scanty (Pl. V, *a*), so that the growth is hard and firm—*Scirrhus* or hard carcinoma. Sometimes the epithelioid cells are not only scanty, but prone to perish early (Pl. V, *b*). Some use the term *Atrophic Carcinoma* for such tumours.

2nd. Those in which the fibrous meshwork is in smaller proportion, and the epithelial contents are plentiful, making large collections of cells, but with no evident approach in the form of these collections to the shapes of gland-acini, and no evident resemblance of the component cells, either to the columnar epithelium of mucous glands or the squamous epithelium of cuticle—*Soft* or *Medullary Carcinoma*. This kind occurs especially in the soft parenchymatous secreting glands, and the transformation of the glandular tubes or follicles to cancer-alveoli can be seen in all stages in the growing margin of the tumour (see Pl. V, *c*, where a renal tubule, containing a cast, lies beside two cancer alveoli developed from neighbouring tubules).

3rd. A structure essentially such as that last described, but with this difference, that the epithelioid cells have a quantity of mucus between them, which is regarded as arising from a transformation of them. This change to mucus may be carried to such an extreme that scarcely any cellular elements are left (Pl. VI, *a*), while the alveolar meshes in which the mucus is contained become very strikingly visible from their nakedness and the pellucidity of the mucus—*Colloid* or *Alveolar Carcinoma*. A common seat of this is the wall of the alimentary canal, where it may be traced arising from Lieberkühn's follicles (Pl. VI, *b*).

4th. A structure in which the epithelial cells resemble squamous epithelium (Pl. VI, *c*), and form masses which are very like the follicles of cutaneous glands (Pl. VI, *d*, *e*), or occasionally like rudimentary hairs; the tubular and bulbous forms may, however, be seen ramifying like the lymphatic vessels of the skin (Pl. VI, *e* to *d*), as if their form were moulded to the lymphatic plexus. In these cancers peculiar bodies are found, composed of flattened cells disposed concentrically so as to form a scaly-walled globe, whose appearance is like the section of an onion, or like a bird's nest (Pl. VI, *e*, *f*); these are so large as often to be visible to the naked eye; when they are numerous and well characterised they are diagnostic (some authors (Billroth) distinguish a variety of this cancer in which the stroma preponderates over the epithelial part, calling it scirrhus of the skin)—*Squamous Epithelial Carcinoma.*

5th. A structure in which the epithelial cells resemble ordinary columnar epithelium, and the structure itself is quite like normal mucous membrane (Pl. VI, *h*), in which it always primarily arises (alimentary canal, especially colon; uterus); the secondary formations which occasionally occur in these cases, in the liver especially, have the same structure, and thus a tissue like the glandular mucous membrane of the colon may be found in the liver—*Cylindrical Epithelial Carcinoma.*

The fourth and fifth varieties are distinguished from the first three as epithelial cancers or epitheliomata. Some authors have used the term cancroid for the fourth variety, as though it were not completely cancerous. These are less likely to infect the viscera than the first two varieties, which are the most infectious of all tumours, though they are very far from being the only kinds of infectious tumours.

When in the first or second variety pigment appears within the cells the name *melano-carcinoma* is applied to the tumour. Other melanotic cancers consist of sarcoma with pigment in its cells; these are called *melano-sarcoma.*

Lymphoma.—The name lymphoma is given to such growths as have a microscopic structure like that of lymphatic glands; in particular, which have a finely reticular meshwork (Pl. VII, *a*), connected with which are some fixed cells at tolerably regular intervals (Pl. VII, *b*), not unlike the fixed cells of connective tissue, but generally larger. Within the meshes of this network are numerous cells, which resemble lymph-cells (Pl. VII, *c*), and hence are also like pus-cells and white blood-cells. The proportion of network to the contained lymphoid cells is variable; sometimes the quantity of fibre is great, and the structure is then like lymph-gland tissue hardened by chronic inflammation. In other cases the proportion of cells becomes very large, while the network grows very delicate and open textured. The fixed stellate cells here appear to multiply, and produce a progeny of the loose movable cells in the meshwork, as if infected by the latter. The whole mass then appears as fine filaments, making bold meshes which are filled with round granular cells, like lymph-cells, but generally larger, and having a large nucleus and many bright nucleoli. These cells at first appear to make up the whole substance, but they easily brush or wash out of the meshes, leaving the network very conspicuous. It will be noticed that the degree of structure here described is very rudimentary. Indeed, in sections of hardened *ante-mortem* blood-clot from within a vein, parts may be found which closely correspond to the description (Pl. VII, *d*). So also, tubercle in the more recently formed outer edge of it, where its texture is very like such portions of blood-clot, has the same texture (Pl. VII, *e*), and hence it is by some classed with lymphoma. Scarlatinous tonsils (Pl. VII, *f*) and typhoid Peyer's patches likewise have lymphoma structure. However, the plan of structure is so meagre that it is not enough to form a bond of union between diseases clinically so remote from each other.

When found in the form of tumours, more properly so called, lymphoma generally takes its rise in the lymphatic glands; those of the neck (Pl. VII, *a*, *b*, *c*) are especially liable to it, then those of the abdomen and of the mediastinum. It is also found in the alimentary canal, especially the small intestine (Pl. VII, *g*) and stomach, and in the spleen, liver, kidney, &c. Formations of a similar structure have been met with in various organs in leukæmia, chiefly in the liver, in the form of small grains of a pale substance.

Lymphoma may prove malignant, that is, infectious to parts around, especially when the cellular elements are very numerous (in which case the tumour is called lympho-sarcoma by Virchow); it then corresponds to a part of what used to be included under the whole name medullary cancer, which, as formerly used, would include also soft sarcoma and soft carcinoma. Indeed, these tumours, when the cell-elements greatly preponderate, become very like each other, if not undistinguishable, as far as their mere structure is concerned.

For the relation of lymphoma to lympho-sarcoma (Pl. VII, *b*) see page 650.

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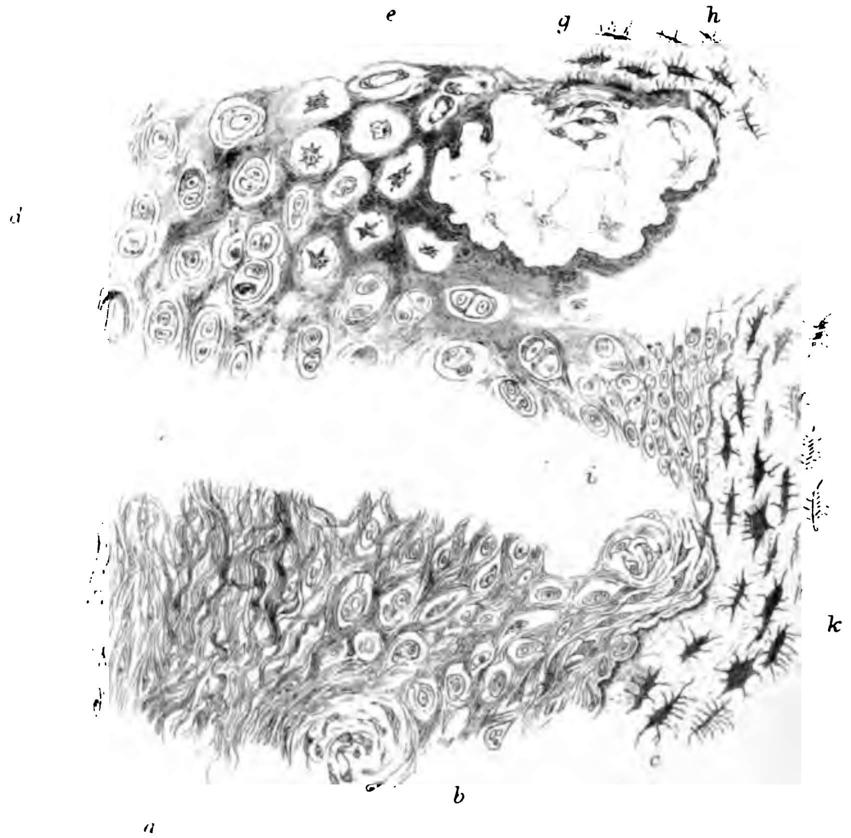
Plate I.



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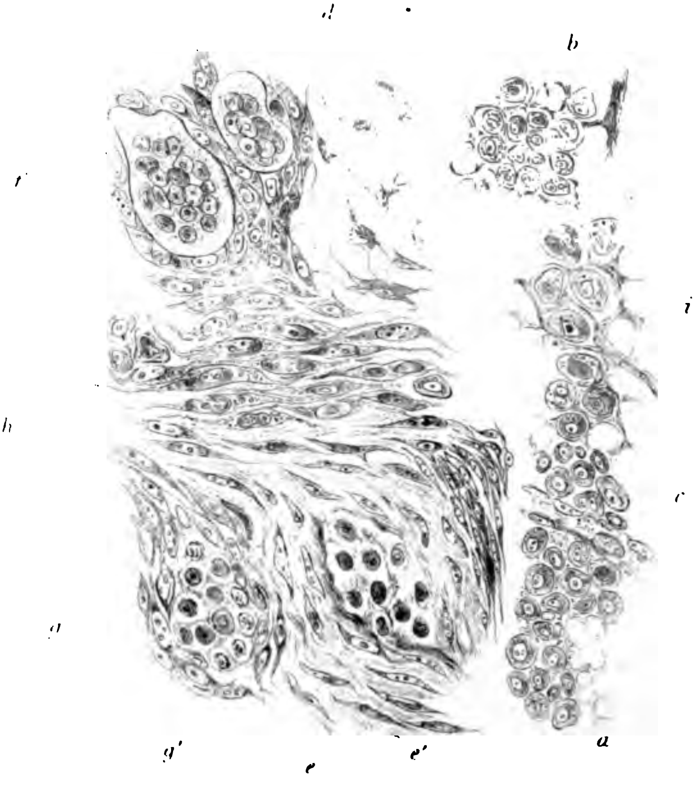

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PLATE IV

a. Adeno-fibroma from lip.
b. Adeno-sarcoma, breast.
c. Adeno-myxoma, breast.
d. Adenoma, breast.
e. Adenoma, subcutaneous, in forearm.

PLATE V

a. Scirrhus from liver, secondary to scirrhus of breast.
b. Atrophic carcinoma, secondary to scirrhus of breast.
c. Medullary carcinoma from kidney; at *c* a renal tubule with a cast, the next tubules changed to carcinoma-areolæ.
d. Spindle-cells in the walls of the areolæ.
e. Round cells in the walls of the areolæ.
f. } Scirrhus from lung, secondary to scirrhus of breast; at *f* the earlier
g. } stage, at *g* the later stage of involution.
h. Scirrhus, cerebellum, secondary to scirrhus of breast.

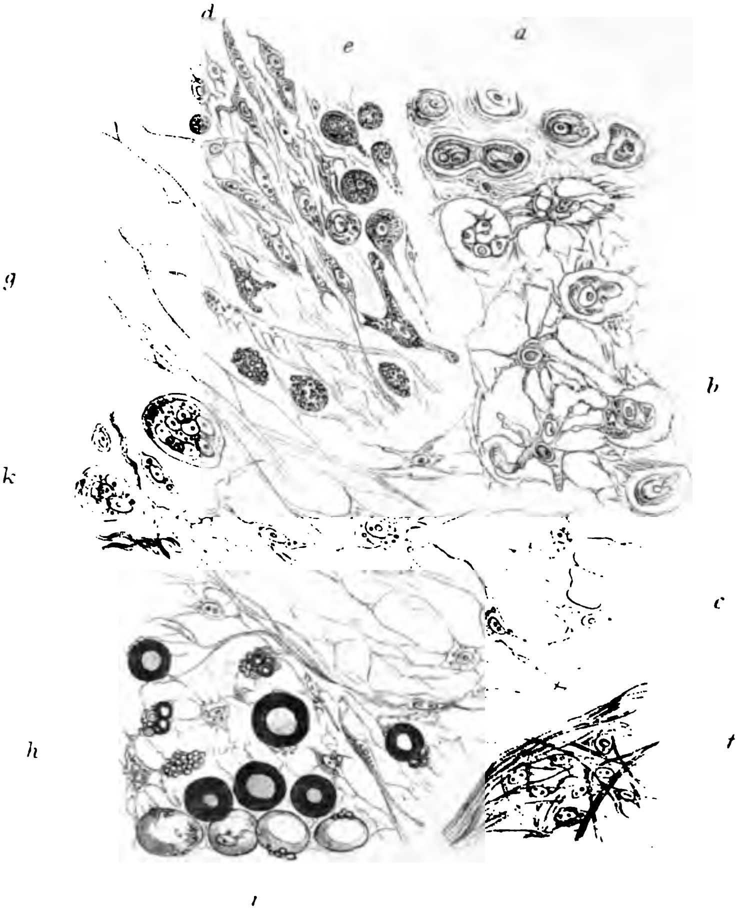
PLATE VI

a. Colloid carcinoma, wall of cæcum.
b. Lieberkühn's follicles near the colloid.
c. Cells of epithelial carcinoma.
e. } Disposition of the epithelial elements in forms resembling acini of
d. } glands, or possibly following the lymphatic channels; at *e* and *f*
f. } "bird's-nest" cells.
g. Richly corpusculated stroma.
h. Cylinder-epithelial carcinoma, sigmoid colon.

PLATE VII

a. Lymphoma, its stroma pencilled out, }
b. Fixed cells connected with the stroma, } from cervical glands.
c. Lymphoid cells filling the meshes, }
d. Portion of ante-mortem blood-clot.
e. Tubercle.
f. Tonsil, scarlatina.
g. Lymphoma, small intestine.

Plate III.



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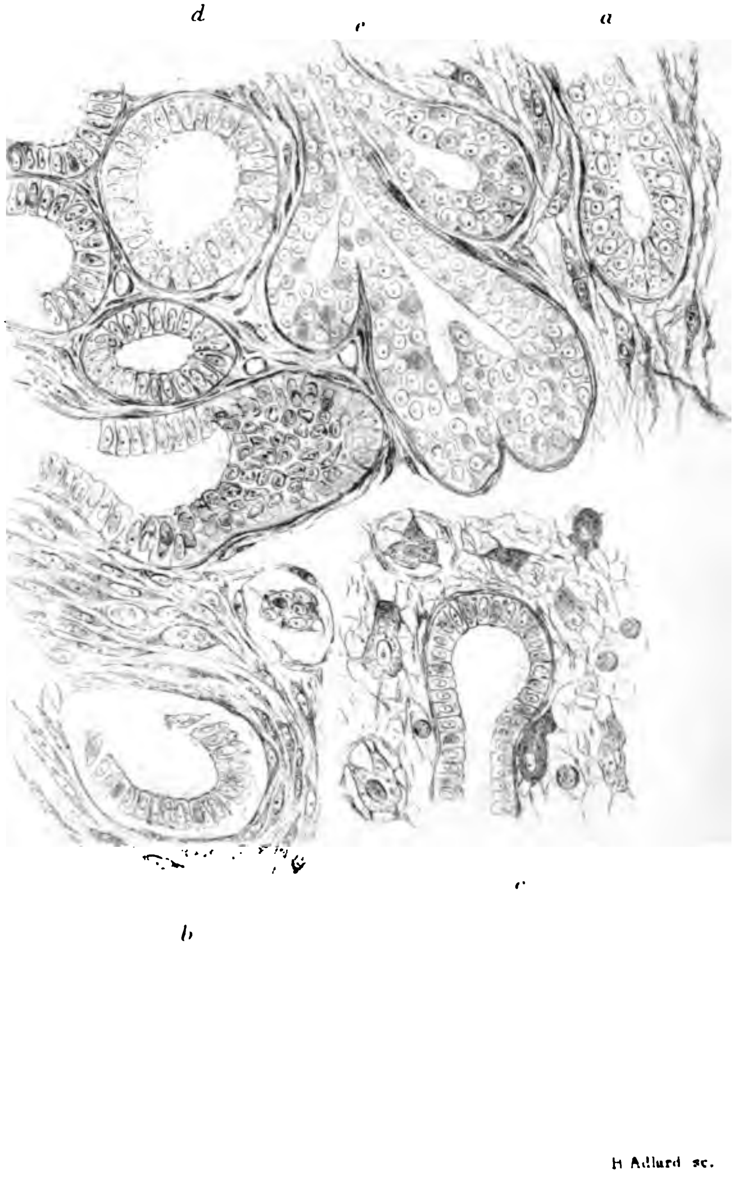

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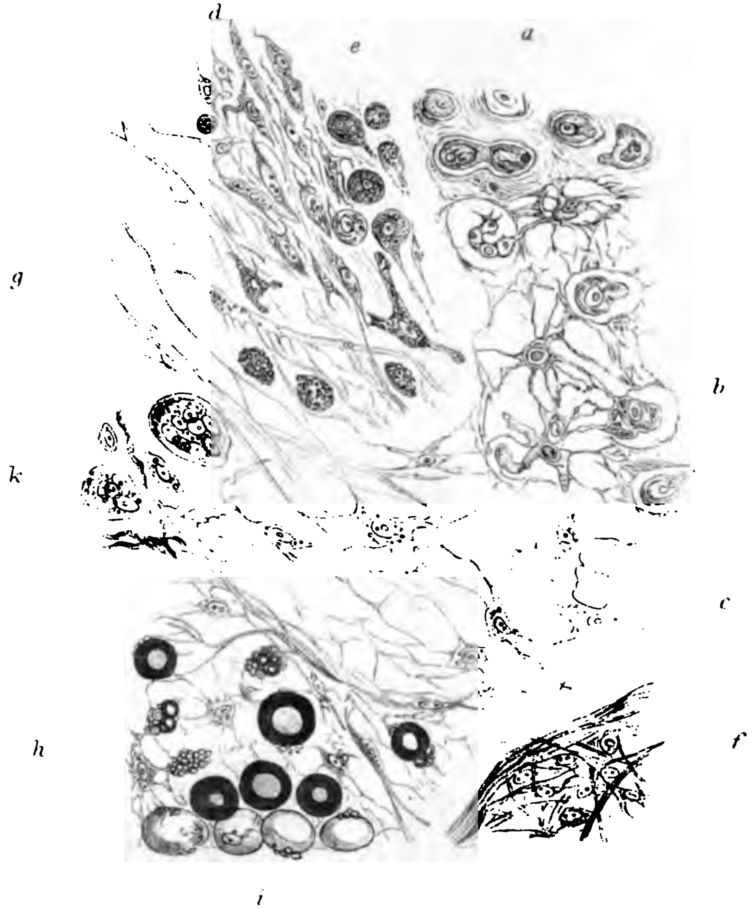

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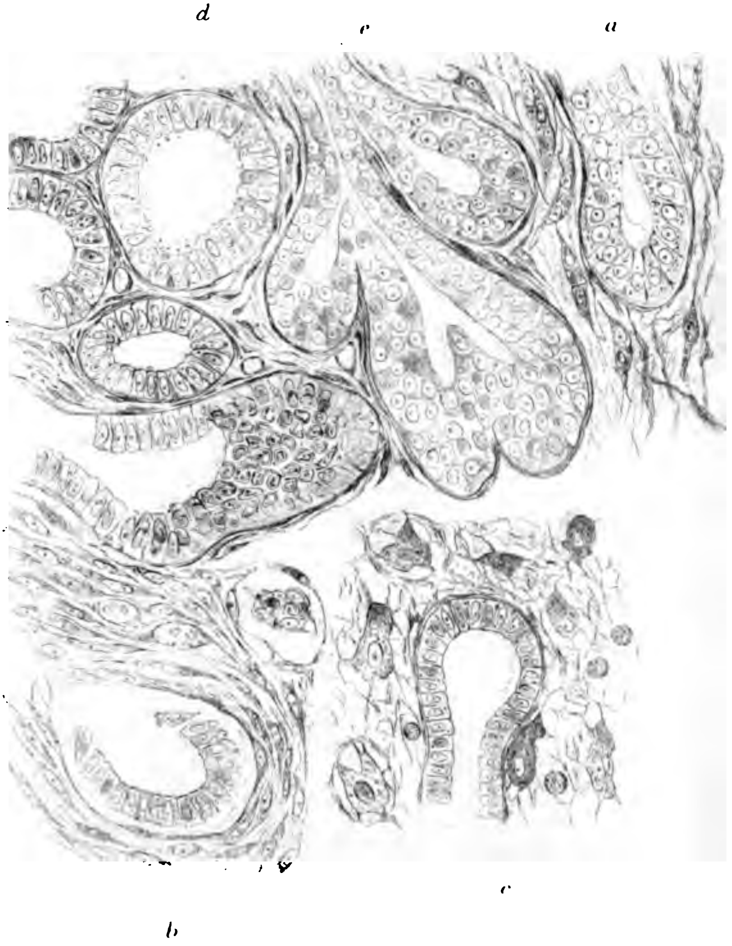

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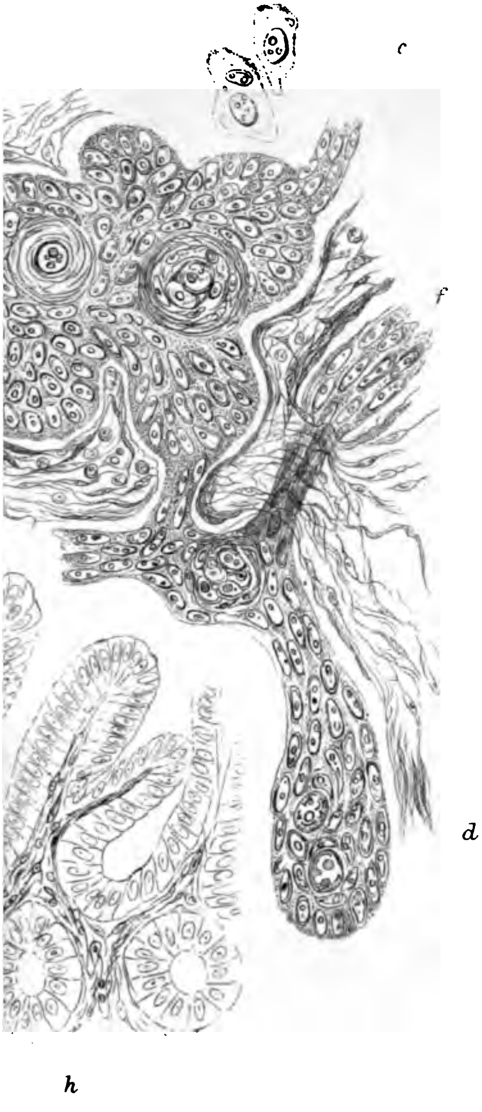

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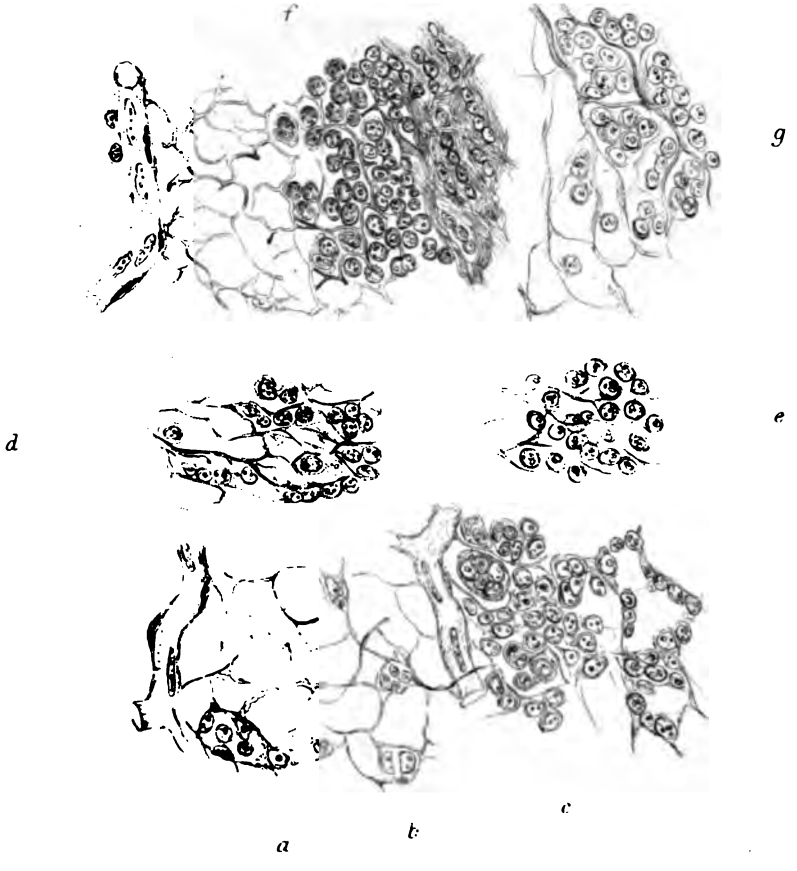

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